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APR 23 2007

PATENT APPLICATION

IN THE UNITED STATES PATENT & TRADEMARK OFFICE

In re application of)
Applicant: Rosskopf et al.)
Title: Methods of Reducing Pest By Use)
Of Iodacetic Acid, Bromoacetic)
Acid, 2-Iodoacetamide, or)
2-Bromoacetamide)
Serial No.: 10/828,802)
Docket No.: 0022.03)
Filed: 4/20/04)
Examiner:)
Group Art Unit: 1616)

PETITION FOR EXTENSION OF TIME

Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

Sir:

Applicants hereby petition for a One month extension of time to respond to the Non-Final Rejection with a mail date of December 28, 2006, thereby extending the period of response to April 28, 2007.

Please charge the \$110.00 fee for said extension to Deposit Account 50-2134.

Respectfully submitted,

4/19/07

Date

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because rabbit heart muscle shows a marked decrease in total acid-soluble phosphorus and organic phosphorus (95). Plasma levels of other important electrolytes are found to be elevated, particularly just before death occurs. Ross reported changes in plasma potassium from control levels of 17 mgm./100 cc. to 25 mgm./100 cc. after poisoning has progressed (52). Others report minor increases in potassium and calcium (95). These electrolyte changes are probably nonspecific and reflect the morbidity of cells weakened by an attack at some other point; similar electrolyte alterations occur under many conditions.

IV. THE INTIMATE MECHANISM OF ACTION

A great deal of illumination has been cast upon the manner in which a drug of a fluoracetate compound brings about the death of an animal. Having been assured that the heart or central nervous system is ultimately disabled by events familiar to pharmacologists, one may properly inquire, "How do these events come about?" Indeed, this is currently the most interesting aspect of the fluoracetate problem.

There is no longer any reason to believe that fluoracetate is in any way pharmacologically analogous to iodoacetate, although this was a natural *a priori* assumption. The behavior of poisoned animals, the chemical character unrelated to iodoacetate and the failure of low concentrations to inhibit significantly any system sensitive to iodoacetate seem sufficient reasons to discard this conception entirely. In addition, even though the chemical nature of fluoracetate suggests no basis for the assumption that fluoride ion might be split off and be the cause of the toxic action of fluoracetate, the actions of the fluoride ion are entirely different from those of fluoracetate. One must of necessity conclude that the fluoracetate ion exerts its action as such and not because of any obvious chemical reactions with it or because of toxic breakdown products.

British workers have succinctly summarized their earlier efforts to identify the systems attacked by fluoracetate as follows: "No enzyme system has been found which is inhibited to any extent by methyl fluoracetate" (96). In a more important communication in 1947 describing work carried on in 1944, Barlett and Barron (12) have reported experiments on the metabolism of animal tissues from which they conclude that, "Fluoracetate probably acts by inhibiting the formation of 'active' acetate (the so-called C_2 compound, which may be an acetyl derivative or an acetate radical)". Because of the importance of this provocative conclusion, it is appropriate to review the observations upon which it is based as well as some experiments which do not justify this conclusion.

4. Tissue Slices, Homogenates and Microorganisms

It was found that 0.001 to 0.2 M fluoracetate decreases the oxygen uptake of tissues. Tissue slices from rats moribund after an LD₅₀ of fluoracetate were found to oxidize acetate 20 to 30% less vigorously than control slices. The degree of inhibition of acetate oxidation could be increased by adding fluoracetate to normal tissues *in vivo*; inhibitions as great as 90% occurred in heart slices after 0.005 M fluoracetate was added. This inhibition might be interpreted as

occurring *per se* with cellular morbidity except for the fact that the inhibition of O_2 uptake of guinea pig brain (which does oxidize acetate and does not convulse) and of rabbit brain (which does not oxidize acetate and does not convulse) were found to be 53% and 11%, respectively.

Experiments were then performed to ascertain the mechanism of acetate blockade and several known routes of pyruvate and acetate metabolism were studied. (1) *Pyruvate* \rightarrow *acetate*: The oxygen consumption of kidney slices in the presence or absence of pyruvate was decreased to about the same extent (60%)

at 0.01 M fluoracetate; yet in the presence of pyruvate, which apparently was oxidized, acetate accumulated. It is apparent that the further oxidation of this acetate had been inhibited. (2) *Acetoacetate* \rightleftharpoons *acetate*: The utilization of acetoacetate by rat kidney slices was completely inhibited by 0.02 M fluoracetate. Conversely, the formation of acetoacetate from acetate was accelerated by fluoracetate and this was still further increased as the proportion of oxygen in the gas phase was increased, reaching 233% of normal in pure oxygen. One may infer from this that acetoacetate is converted in *vivo* to acetate, the further oxidation of which is inhibited so that acetate accumulates as the end-product of the reactions: glucose \rightarrow pyruvate \rightarrow acetate and forces the reaction: acetate \rightleftharpoons acetoacetate. (3) *Pyruvate* \rightarrow *acetate* \rightarrow *succinate*: The formation of succinate from pyruvate was 77% inhibited by 0.02 M fluoracetate. Since none of the individual reactions of the Krebs' tricarboxylic acid cycle was found to be affected by fluoracetate (see also (84) for a statement of disagreement), this may be interpreted to indicate that pyruvate enters the Krebs' cycle in large part through acetate and not directly. (4) *Other reactions*: Pyruvate and lactate formed by deamination of alanine accumulate in rat kidney slices in the presence of fluoracetate, a fact that might be readily predicted if it were established that oxidation of pyruvate through acetate is inhibited. Glucose metabolism is directed toward lactate (aerobic glycolysis) to the extent of nearly one half, but the anaerobic utilization of glucose is completely unaffected in kidney slices. Aerobic oxidative synthesis of carbohydrate from pyruvate and acetate is markedly inhibited by fluoracetate; this fact suggests that, as the entry of acetate into Krebs' cycle is inhibited, this cycle may be a pathway for the synthesis of carbohydrate from pyruvate and acetate. Anaerobic conversion of pyruvate to lactate and acetate was not affected (see also 26). (5) *Acetylation of foreign substances*: The acetylation of sulfanilamide and of p-aminobenzoic acid by rabbit liver slices was increased by 0.02 M fluoracetate. (This has been confirmed in rats and rabbits (37, 123).) The chemical reaction of acetylation is not affected; formation of acetylcholine from choline in the presence of glucose or pyruvate is not affected by 0.02 M fluoracetate (see also 85). Therefore, it may be assumed that the increased acetylation of foreign amines is the result of an inhibition of acetate metabolism in consequence of which more acetate becomes available for acetylations.

Having been thus provided with a basis for the concept that fluoracetate interferes with the oxidation of acetate in various animal tissues, Kalnitsky and Barron (77) studied the details of the phenomenon in baker's yeast and bacteria.

The oxidation of acetate by baker's yeast was 95% inhibited by 0.001 M fluoroacetate (30% by 0.0001 M), only 5% by 0.001 M bromoacetate and not at all by 0.001 M chloro- and iodoacetate; the specificity of the reactions involved thus demonstrated. Fluorobutyrate and fluorocrotonate (0.001 and 0.003 M) had no inhibitory action at all. The oxygen uptake of yeast suspensions nearly completely inhibited by fluoroacetate added 15 minutes before acetate and practically unaffected when the two were added together. Appreciable reversal of such fluoroacetate-induced inhibition could be obtained by adding higher concentrations of acetate (0.1 M). Additional evidence for the specificity of the inhibition was obtained when it was found that acetate oxidation, especially washed yeast, was completely inhibited by 0.00075 M fluoroacetate while pyruvate oxidation was only 79% inhibited.

By the use of a different approach, it was reasoned that if fluoroacetate is a specific inhibitor of acetate oxidation there should be no immediate inhibition of the O_2 uptake associated with the oxidation of ethanol to acetate through acetaldehyde. This was found to be the case; oxidation of ethanol by baker's yeast in the presence of 0.01 M fluoroacetate progressed exactly as in the absence of fluoroacetate until the accumulation of unoxidized acetate affected the rate of oxidation of ethanol to acetate. Less complete inhibition of acetate oxidation produces less block of ethanol oxidation; Black and Hutchens (15) found that 0.001 M fluoroacetate did not completely prevent the continuation of ethanol oxidation through acetate.

The anaerobic dissimilation of pyruvate to acetate and formate by *Escherichia coli* was unaffected by 0.01 M fluoroacetate although the oxidation of pyruvate to acetate by this organism was definitely inhibited. *Neisseria gonorrhoeae* did not dissipilate pyruvate but does oxidize it directly to acetate. This reaction is 40% inhibited by 0.01 M fluoroacetate, 26% by 0.02 M acetate and 52% by both together. These experiments add emphasis to the view that inhibition of pyruvate oxidation by fluoroacetate is due to the accumulation of acetate, since even acetate alone is slightly inhibitory.

Quite different results were obtained when another microorganism, *Corynebacterium cretininovans*, was studied. An increase in the endogenous respiration of this organism was produced by both fluoroacetate and fluorobutyrate. It was suggested that this is the result of diversion of cellular metabolism by fluoroacetate toward oxidative pathways in a manner similar to that produced by concentrations of cyanide and azide. Although acetate oxidation by this organism was completely inhibited by fluoroacetate, fluorobutyrate had no effect at all.

As was mentioned, the greatest inhibition of acetate oxidation (as measured by decreased O_2 uptake) by fluoroacetate occurred when fluoroacetate was added to the yeast some minutes prior to the addition of the acetate substrate. After two hours the inhibition apparently decreased considerably (as measured by increased O_2 uptake), a fact which Kalnitsky and Barron interpreted as the indirect result of the slow accumulation of citrate and its increasing movement into the carboxylic acid cycle. The same characteristic decrease of inhibition was noted

in the growth curves of *Tetrahymena geleii* in a glucose but not in an acetate medium (48). A further study of the effect of fluoroacetate upon citrate formation was later made by Kalnitsky (75, 76). Using rabbit kidney cortex homogenate, he found that not only fluoroacetate but barium and magnesium salts well appear to increase the formation of citrate from oxaloacetate. The effect of barium and magnesium is the result of inhibition of citrate utilization. On the other hand, the slight inhibition of citrate utilization (at fumarate? (84); v.i.) produced by high concentrations of fluoroacetate can only account for about 10% of the accumulation of citrate in the presence of fluoroacetate. It was concluded that the increased citrate content might be the result of the inhibition of acetate oxidation reflected through a series of reversible reactions in a manner analogous to the effect of malonate on pyruvate oxidation.

Another approach to the problem of the greater inhibition of fluoroacetate on acetate oxidation by yeast when the fluoroacetate was added before the acetate was made by Black and Hutchens (15). Working in different laboratories, they confirmed the general fact that a more prolonged inhibition of acetate oxidation is obtained by allowing a longer period to elapse between the addition of the inhibitor and the substrate. They suggest that this is similar to the delay noted by Lynen (91) in starved yeast before acetate oxidation becomes vigorous and that it is the result of cellular depletion of certain substances found by Lynen to be essential for acetate oxidation in yeast. Ethanol was found to be particularly efficacious in accelerating the oxidation of acetate by yeast either untreated or pretreated with fluoroacetate.

When calculations of the oxygen consumption of yeast were made using the initial rate after equilibrium has been reached, Black and Hutchens found that pyruvate oxidation is more sensitive to fluoroacetate than is acetate oxidation. Because the delay in oxidizing pyruvate is less than for acetate, these workers decided that the earlier conclusion of Kalnitsky and Barron that acetate oxidation is more sensitive than pyruvate was the result of an error in technic. Hutchens, McMahon and Podolak (69) have recently reported that the inhibition by fluoroacetate salts of pyruvate-induced oxidations in yeast depends on the pH of the medium. However, inhibition of acetate-induced oxidations in yeast and *Chilomonas paramecium* is independent of pH. While this may have been one of the actual causes of differences in opinion concerning the specificity of fluoroacetate-induced inhibitions in yeast metabolism, Hutchens *et al.* find pyruvate oxidation much more sensitive than acetate oxidation in the case of *Chilomonas*. Although it is very hazardous to change species in the middle of an argument about fluoroacetate, it can be pointed out in support of this that Bueding (26) noted that in the filarial worm, *Liomyosoides carinii*, pyruvate oxidation is very much more sensitive than acetate. In addition, he stated, "No evidence has been obtained that fluoroacetate inhibits the respiration of the filariae because of a competitive inhibition of acetate oxidation." Fluoroacetate (0.001-0.004 M), while producing a decrease in the total respiration and motility of the organism, actually produced an accumulation of pyruvate and a decrease in the formation of acetate from glucose. Although in this study he has rigorously tested these

conclusions, Bueding also demonstrated that the metabolic characteristics of *L. carinii* are unique in that they differ from those of other helminths and, indeed, from those of most other invertebrates.

The anthropocentric may draw more comfort, therefore, from the results of a later study by Kalnitsky and Barron (78) on the effects of fluoroacetate and fluorobutyrate on fatty acid and glucose oxidation in kidney homogenates. Homogenates of rabbit kidney cortex oxidize acetic and many other fatty acids vigorously. The oxidation of acetate was immediate, and practically completely inhibited by 0.001 M fluoroacetate and fluorobutyrate. This is in contrast to the total lack of effect of fluorobutyrate on acetate metabolism in yeast. This discrepancy was shown not to be the result of conversion of fluorobutyrate to fluoroacetate by kidney. Two other sharp differences between yeast and mammalian tissue were noted. The apparent release of the fluoroacetate-induced inhibition of yeast metabolism with time does not occur in kidney suspensions nor does ethanol have the least effect on the degree of acetate oxidation. Fluorobutyrate proved to be a more potent inhibitor of butyrate oxidation than did fluoracetate; 0.00005 M fluorobutyrate inhibited butyrate oxidation 86% while at the same concentration fluoroacetate produced only 32% inhibition. Oxidation of higher fatty acids was also inhibited to varying extents by both fluoroacids.

In contradistinction to the case in yeast where it developed slowly, glucose oxidation was rapidly inhibited by fluoroacetate in kidney homogenates, as was also that of acetate. However, as in their previous studies, they found that pyruvate oxidation was not inhibited until a considerable portion (20%) of the added pyruvate had been oxidized. It appears that the specificity of fluoracetate inhibition described for yeast is not to be found in mammalian tissue, in this case, rabbit kidney. It is interesting that fluorobutyrate is a more effective inhibitor of butyrate oxidation than is fluoroacetate. One can only speculate at this time about the effects of appropriately fluorinated higher fatty acids.

By actual analysis for acetate, Colowick, Berger, Stein and Cori (41) found that rabbit kidney cortex homogenates removed about 35% less acetate after addition of 0.005 M fluoroacetate. They add the new information that the extinction of various metabolites is inhibited by high concentrations of fluoroacetate (0.05 M) to varying degrees. Glucose is most sensitive, the extra oxygen uptake being inhibited 68%, and phospho-enol pyruvate is approximately the same, the inhibition being 65%. Unfortunately, acetate does not appear to have been tested. The effect of fumarate was found to be inhibited 53% but other metabolites and components of the tricarboxylic acid cycle were relatively little affected.

The curious sensitivity of fumarate-induced extra oxygen consumption does not appear to be entirely coincidental for Boyarski, Postel, Rosenblatt and Gerard (18) found it to be specifically effective in preventing the decrease in the action potential of methyl fluoroacetate-poisoned frog nerve. On the other hand, actually increased the toxicity of sodium fluoroacetate to intact rabbits (36). These matters require considerable clarification. Lisbecq and Peters (84) have reported recently that studies with centrifuged, homogenized guinea pig kidney

and pigeon brain preparations indicate the possibility that a "fluoro-C₂ active fragment" may be formed which enters the tricarboxylic acid cycle and becomes an inhibitor of this cycle. They found, as did Kalnitsky (76), that citrate accumulates during poisoning *in vitro* while acetate does not. The relation of results of many *in vitro* studies to the events occurring *in vivo* is difficult to establish. It is possible that more physiological preparations could prove more useful.

B. Working Muscles

The use of an actively functioning, normally organized preparation such as the frog sartorius muscle has led to some additional and slightly different information. Colowick, Berger, Stein and Cori (41) found that the oxidative resynthesis of phosphocreatine by frog sartorius following one minute of tetanic stimulation was depressed as much as 40% by previously soaking the muscle for 1 hour in 0.005 M methyl fluoroacetate. These findings were explained as the indirect result of depressed tissue respiration, for the simultaneous oxygen uptake of these muscles following stimulation was decreased from an average 100% increase to only 35% increase. Essentially the same results were obtained when dinitrophenol was used to increase basal oxygen consumption; for 0.005 M sodium fluoroacetate inhibited the increase about 70%.

Similar results were obtained when caffeine was used by Clarke and Riker (39) to stimulate the frog sartorius, the excess oxygen consumption of muscles contracting under such circumstances being decreased by methyl fluoroacetate. The respiration of resting muscles is not affected significantly at similar concentrations of methyl fluoroacetate. The oxidative recovery heat which normally follows a single maximal twitch was abolished by 0.001 M methyl fluoroacetate or 0.01 M sodium fluoroacetate, yet the muscles continued to contract, exactly the reverse of the situation with iodoacetate where an increasing oxidative recovery heat production may accompany contractile failure. The depression of the activity oxygen consumption of these muscles induced by fluoroacetate can be abolished completely by 0.01 M acetate, glycerol monoacetate, pyruvate and iodoacetate. Ketoglutarate, fumarate, malate and succinate were less effective, and glucose and lactate were without any significant effect.

In a continuation of this study, Clarke and Riker have recently found that, unlike iodoacetate, fluoroacetate does not inhibit anaerobic glycolysis and, under the conditions of their experiments with frog muscle, the rate of glycolysis is gradually increased. Because the aerobic accumulation of lactate that normally results from muscle activity is significantly less in the presence of fluoroacetate and since lactate formation is not inhibited by fluoroacetate, it appears that lactate must be metabolized in the poisoned muscle. The explanation of these results is to be found in the fact that, in this preparation at least, the action of fluoroacetate appears to be to inhibit oxidative carbohydrate breakdown; as a consequence, the anaerobic carbohydrate breakdown which occurs in activity is enhanced, and this results in a rapid turnover of lactate without its accumulation. It is evident that this will provide sufficient energy to permit muscular contraction.

Three independent studies of the effect of fluoroacetate upon the spontaneous contractility of isolated upper segments of rabbit small intestine have been made.

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MONOFLUOROACETIC ACID AND RELATED COMPOUNDS

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otherwise substrate-free preparations of the type described by Furchtgott and Shorr (55) led Furchtgott to study the interrelations of fluoroacetate and acetate on this preparation. Experiments performed during 1946 by Furchtgott (54) indicated that glucose could supply energy for contraction of intestinal smooth muscle in the presence of fluoroacetate under either aerobic or anaerobic conditions, although acetate could not. Fluoroacetate poisoning was irreversible when carried out under aerobic conditions. However, if fluoroacetate was added during a period of anoxia, allowed to remain in the muscle chamber for over 30 minutes, and then washed out before the restoration of oxygen to the muscle, there was no toxic effect.

Later, Farah, West and Angel (50), upon examining this system found that both glucose and acetate were effective antagonists to fluoroacetate, and that there are considerable differences in the character of the response of the gut to fluoroacetate in the presence of these substrates. They have shown that although fluoroacetate depresses contractility more rapidly when glucose is the sole substrate than when acetate alone is present, the percentiles decrease in amplitude of contraction at a given concentration of fluoroacetate is greater in the presence of acetate than when glucose is the substrate. There is a direct relation between (a) the time required for 0.0008 M sodium fluoroacetate to produce 95 to 100% inhibition of contraction and (b) the concentration of sodium acetate present in the bath, the higher the concentration of acetate the longer the time required for this inhibition to result. Butyrate and pyruvate, when they are the sole substrates, are not detectably different from acetate with respect to the percentiles reduction in contraction amplitude produced by a given concentration of fluoroacetate.

Although regular contractions in glucose are stopped by high concentrations of fluoroacetate, there remains an irregular, high amplitude "fluoroacetate-resistant" contraction when glucose is present with or without other substrates. This phenomenon is seldom or never seen when acetate alone is the substrate. It was noted by all groups which investigated the problem. Farah *et al.* (50) have found that only mannose acts like glucose whereas galactose, fructose, pyruvate, butyrate, caproate, caprylate, succinate, fumarate and α -ketoglutarate are, likewise, unable to support these contractions. These contractions are not abolished by anaerobiosis (N_2 , cyanide) or by malonate, although azide and iodacetate are effective in abolishing them. It has been known for some time, and mannose as a source of contraction energy during anaerobiosis. They feel that it is possible that energy for the fluoroacetate resistant contractions may be obtained from anaerobic glycolytic pathways.

That glucose is more effective than acetate in maintaining motility of the segments in the presence of high concentrations of fluoroacetate was confirmed by Weeks and Chenoweth (132). When intestinal strips are allowed to contract

in exhaustion in Krebs-Henseleit solution in the absence of substrate, the normal stimulatory action of added 0.005 M sodium acetate is prevented by addition of 0.0032 M sodium fluoroacetate 5 minutes before the sodium acetate. Whereas subsequent addition of 0.005 M glucose is still effective. Although they found that glycerol monoacetate is a very effective antagonist to fluoroacetate *in vivo* and sodium acetate is definitely not, in the isolated intestinal segment preparation sodium acetate is nearly five times as effective as glycerol monoacetate against fluoroacetate. Other experiments demonstrated that glucose-induced contractions, after fluoroacetate inhibition (0.01 M) in acetate, were of the same amplitude (approximately 30% of control values) as the contractions which persisted when the same concentration of fluoroacetate was added to muscles with a glucose substrate. These various observations can probably be explained by assuming, and there appears to be good reason to do so, that intestinal muscle under these circumstances obtains energy for contraction from at least two sources: (1) the breakdown of glucose which can occur anaerobically and (2) reactions of the tricarboxylic acid cycle into which acetate enters. When acetate, as the sole available substrate, is blocked from entry into the cycle by high concentrations of fluoroacetate, contraction ceases; in contrast, low concentrations of fluoroacetate are unable to produce a block in the presence of excess acetate. In the presence of glucose, energy for contraction is still available despite the blockade of acetate produced by fluoroacetate. This blockade may be increased until only the fluoroacetate, anaerobiosis-resistant contractions remain.

C. Working Nervous Tissue

According to Shanes and Brown (117), the preservation of the resting potential of frog nerve depends upon formation of pyruvate by the glycolytic cycle and the subsequent aerobic metabolism of this substrate. Because 0.01 M methyl fluoroacetate interferes with the redevelopment of the resting potential of nerve in oxygen following a period of anoxia, Shanes (116) felt that this was sufficient to suggest an interference with pyruvate metabolism. Conversely, the simultaneous addition of methyl fluoroacetate and sodium pyruvate to nerve in oxygen maintains a higher resting potential than when pyruvate is omitted, a fact which suggests a beneficial effect of added pyruvate not noted with acetate. During poisoning, he found that the threshold of excitability of frog sciatic nerve to condenser discharges steadily increased.

A series of studies has recently been published which has revealed several new facts about the action of fluoroacetate (17, 18, 22, 23, 45, 98). The sodium salt of fluoroacetic acid is nearly without action on frog nerve or brain *in vitro* although methyl fluoroacetate has several actions. Thus, the ester decreases the resting potential of frog sciatic nerve and reduces conduction velocity by a process of blocking conduction in fibers; the threshold of the larger fibers is raised before that of the smaller fibers at concentrations of 0.005 M. The respiration of such nerves is decreased to 20% of normal. Sodium fumarate added before, up to 15 minutes after, the methyl fluoroacetate protects against the action

potential changes in a 2:1 molar ratio, but the respiration of the nerve still be 50% decreased. Succinate is equally effective in a 5:1 molar ratio, ethanol, acetate, pyruvate, α -keto-glutarate and glucose are ineffective. Although their data have not yet been fully reported, Doty and Gerard have found that methyl fluoracetate will depress the resting oxygen consumption of frog nerve as described, but that the increased oxygen consumption which occurs on stimulation is not affected. Thus, with 0.001 M methyl fluoracetate the resting Q_{O_2} is decreased by 25% whereas the activity increase is unaffected. The absence of respiration, non-conducting elements in a nerve trunk must be considered when interpreting these results.

In Gerard's isolated frog brain preparation, sodium fluoroacetate is ineffective whereas methyl fluoroacetate inhibits respiration 45% at 0.012 M (23). At this level the brain potentials are decreased about 50%. At 0.01 M methyl fluoroacetate there may also be a 50% decrease in cholinesterase activity. On preparation the beneficial effects of fumarate are again seen, but they are rather somewhat less specific by observations that fumarate protects to some extent against di-isopropyl fluorophosphate and that under certain circumstances little as 0.000,001 M sodium fumarate can itself induce bizarre electrical changes in the brain.

The inactivity of sodium fluoroacetate does not appear to be solely the result of inability to penetrate cells, for it is inactive on rat brain or dog nerve homogenates in which cells are disrupted. Malic dehydrogenase of rat brain is sensitive to methyl fluoroacetate (20% inhibited at 0.001 M), but in general dehydrogenases are not much affected by fluoroacetate (98).

D. Isolated Perfused Hearts

By the use of a recirculating system for perfusing isolated hearts through coronary arteries over long periods with bacteria-free Ringer's solutions (22) it has been found that concentrations of methyl or sodium fluoroacetate comparable to those calculated to exist in rabbits or rhesus monkeys poisoned with LD₁₀₀ cause a gradual decline in the amplitude of contraction, occasionally fibrillans and rarely, if ever, fibrillation (28). The relative sensitivity of rabbits and monkeys to fluoroacetate is also manifested in their isolated hearts. Finally the same degree of cardiac incompetence was produced by methyl fluoroacetate acting over a two-hour period in a concentration of 0.00001 M (0.5 mgm./L) on the isolated rabbit heart as by an intravenous dose of 0.5 mgm./kgm. of the intact animal (equivalent to about 0.6 mgm./L of body water). In the intact monkey heart, 0.001 M fluoroacetate in the perfusion fluid produced effects which correspond roughly to those of an intravenous dose of 5 mgm./kgm.

The substitution of sodium acetate for the glucose of the perfusate in rabbit and monkey hearts effectively maintained contraction and exerted the same protection against the effect of added fluoroacetate (33). In some instances protection was definite when the molar ratio of acetate to fluoroacetate

E. Mechanism Studies in Intact Animals; Antidotes

Because humans accidentally or wilfully ingest rat poisons, there can be no doubt of the desirability of an effective antidote to fluoroacetate. The studies so described do not offer much hope that any highly effective treatment of well-established fluoroacetate poisoning will be found. Indeed, most investigators have

understanding of the mechanism of action of the poison.

It has been mentioned that sodium acetate, although it is the most likely antidote, is not an effective antidote or prophylactic in rabbits poisoned with fluoroacetate. However, Tourtelotte and Coon (125) have found that in mice, at least, sodium acetate (2 to 3 gram/kgm.) will protect against sodium fluoro-

acetate. Ethanol, which may be considered simply a source of acetate *in vivo* or, more complexly, a catalyst of the Krebs' cycle, is also effective (1.6 gram/kgm.). Ethanol and acetate together are distinctly more than twice as effective than either alone, a fact which suggests a synergistic effect.

Buthman *et al.* (70) have reported more fully on the effectiveness of ethanol or with sodium acetate in protecting mice against fluoroacetate. Ethanol is also effective in rabbits, and to a lesser extent in guinea pigs, but not at all in dogs or not mice. Although there is no doubt that the judicious administration of barbiturates (bearing in mind the prolongation of sleeping time described in U.G. c.) will control convulsions induced by fluoroacetate in dogs (52, 70, 71), animals which survived in this laboratory manifested changes characteristic of critical damage. It would thus appear that, although overt convulsions are prevented, the pathological pattern of fluoroacetate poisoning has been unaf-

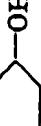
fel. The effectiveness of sodium acetate *in vitro*, despite a generally unfavorable response to it *in vivo*, led to a search for other sources of C₂ moieties. One of the most promising has been glycerol monooacetate which has protected rats, rabbits, and rhesus monkeys against fluoroacetate (36, 37). Equimolar doses of

glycerol monoacetate and ethanol, given to rhesus monkeys after fluoracetate poisoning had progressed, were strikingly different in effectiveness, ethanol apparently being of no value although its effectiveness in mice and rabbits has been confirmed. Several other such compounds have been found which exert protective action against either fluoroacetate or fluorocrotonate.

The simultaneous administration of large amounts of insulin and glucose often effective in dogs and rabbits, although neither substance is effective alone (37). (The forcing of glucose is also protective against anoxic anoxia (38).) Anoxia caused by methemoglobinemia (approximately 70%) produced either by sodium nitrite or p-aminacetophenone is a very effective antidote or prophylactic for fluoroacetate poisoning in those species in which it can be induced, *e.g.*, mice and dogs (37). McNamara (97) has recently found that physiological sodium chloride solution exerts a definite protective effect against fluoroacetate poisoning in rabbits, a fact which may account, in part at least, for the heterogeneous character of some of the substances that appear to exert a moderate protective action. It serves no useful purpose to list in detail all those substances which have proven ineffective; but, in general, salts of fatty acids, anticonvulsants, vitamins and most metabolic intermediates are without effect. Potent antifibrillatory, autonomic and cardiac drugs are generally of no therapeutic value; they may act differently after fluoroacetate (*e.g.*, 50).

V. MISCELLANEOUS FLUORINATED COMPOUNDS

Several familiar compounds in which fluorine has replaced hydrogen or chlorine have been prepared and are of some interest here. Although 2,3-difluoro succinic acid (80), HOOC—CHF—CHF—COOH, appears to inhibit succinic dehydrogenase completely in very low concentrations, it is of singularly low toxicity (37). When fluorine is substituted for chlorine in sesqui-H, a very potent analog of mustard gas, the resulting compound F—CH₂CH₂—S—CH₂CH₂—S—CH₂CH₂—F, is nontoxic with neither vesicant nor fluoroacetate-like activity (88). This suggests that the body is unable to rupture the C-S link in this compound to obtain fluoroacetate and adds emphasis to the fact that the vesicant action of the mustards is dependent upon reactive halogens. Other compounds in which fluorine has been substituted for an hydrogen atom, such as di-(2-fluoroethyl)fluorophosphate or triethyl lead fluoroacetate, combine some of the characteristic activity of the parent compound with that of fluoroacetate (109). Mention should be made of the potent antithyroid activity reported by Litzka (87) in

3-fluorotyrosine HO——CH₂CH(NH₂)COOH. This activity may be due to the presence of the fluorine atom at the para position of the tyrosine ring. The presence of the fluorine atom at the para position of the tyrosine ring is likely to be the cause of the high toxicity (LD₅₀, 12.5 mgm./kgm.), since 1- or 4-fluorotyrosine acids are virtually nontoxic (79, 86). Boyer, Evans and Phillips (19) were unable to demonstrate an effect on the basal metabolic rate of rats, although they confirmed the toxicity of the compound. These compounds have been included because they give some indication of the way in which fluorine atoms in place of

VI. DISCUSSION

Nearly all the original data on monofluorinated fatty acids so far available in the open literature and much of that in the classified literature have been presented in this review. There appears to be a plethora of information on less important points but a dearth of incontrovertible data on the more important conclusions. What interpretation can be given the facts now available? It is clear that the substitution of one fluorine atom for one hydrogen atom in the terminal position of a straight chain fatty acid containing an even number of carbon atoms, to cite the best-defined series. Thus 2-monofluoroacetic acid is active, 4-monofluoropropionic acid is inactive and 2-monofluorobutyric acid is very active, although 2-monofluorobutyric acid is inactive, and so on. Substitution of 10 atoms or groups other than one fluorine atom on the terminal carbon atom does not result in characteristic activity. More complex compounds containing suitable grouping, for example, esters of 2-monofluoroacetic acid or 2-fluorobutanol, exert a typical pharmacological action if they can be broken down in the organism to yield fluoroacetic acid. This principle has been applied in a few less simple cases to indicate whether or not the body can rupture certain linkages, the C-S bonds in the chain F—C—C—S—C—C—S—O—C—F being an example. It appears to be a technic of some value, although open to criticism on the grounds that such a compound might be a specific inhibitor *per se*, should not prove to be inert (for instance, fluoroacetyl salicylic acid).

The toxicity of suitably substituted even-numbered fatty acids in contrast to the inertness of odd-numbered homologs is superficially further evidence for oxidation of fatty acids, the assumption being that the even-numbered chains are broken down to the toxic fluoroacetic acid. Then, in order to account for the much greater toxicity (and the relative differences in various species) of the longer chains as compared to fluoroacetic acid on a mole per kilogram basis, it is necessary to assume that (a) they penetrate cells much more efficiently and/or (b) that release of fluoroacetate as an "active" F-C₂ radical occurs, preferably in the area where it could do the most harm. To the contrary, the quantitative and qualitative differences between the actions of the fluorinated higher fatty acids and the shortest active acid suggest most strongly that they act at separate sites. Evidence has been presented that fluorobutyrate, for example, is much more active inhibitor of butyrate oxidation in mammalian tissue than is fluoroacetate, while in yeast fluorobutyrate does not inhibit acetate oxidation at all although fluoroacetate actively does so. Under at least one set of circumstances it has been shown that fluorobutyrate does not break down to fluoroacetate. In addition, the gross pharmacological effects of fluorobutyrate differ qualitatively from those of fluoroacetate, although admittedly the difference requires

sharper definition. While it is impossible on the basis of available information to exclude the possibility of β -oxidation of fluorinated higher fatty acids, fluorooacetate, it appears probable that they exert their toxic action in part, at least, by interfering with the metabolism of the corresponding non-fluorinated fatty acid in the cell. It is thus equally easy to dispose of the inactivity of the numbered fluorocarboxids, for it is generally uncommon to find the non-fluorinated homolog important in the main line of fatty acid oxidation.⁴

Certain inactive compounds shown in Table III might be used to

weight to the hypothesis of β -oxidation to fluoroacetate. For example, $\text{F}-\text{CH}_2-\text{C}(\text{O})-\text{CH}_2\text{COOH}$ is pharmacologically inactive, and on structural grounds

not be expected to undergo β -oxidation to fluoroacetic acid. Similarly, derivatives of fluorobutyric acid in which carbon atoms 2 and 3 are part of a cyclic structure are inactive, and are not susceptible to β -oxidation. Although the inactivity of these compounds adds support to the idea that β -oxidation of higher ω -fluorocarboxids is necessary for activity, it is equally possible to argue that because of steric factors these biologically abnormal compounds never have an opportunity to inhibit butyrate oxidation inasmuch as they are kept from entering a reactive center by their structural deformity and for that reason are pharmacologically inert.

In this case the assumption of "entrance into reactive centers" is based entirely upon the remarkable similarity (see Table I) of the FCH_2 - and HCH_2 -radicals, the chief differences being that the fluorine atom is about twice the size of the hydrogen atom and is bound more securely to the carbon atom. (Were there physical-chemical differences at all there would seem to be no reason to expect any pharmacological differences.) If it be assumed that in the process of metabolism fatty acids fit some sort of matrix which requires entry of the terminal methyl group, it is easy to visualize the arrival of a fluorinated methyl group which fits effectively; but, perhaps because of the greater hold the carbon hydrogen bonding has upon each other and because of fluorine's propensity for hydrogen bonding, it cannot readily be dislodged. In such a case as fluorooacetic acid the prior administration of substances which release large amounts of acid in the cell could be expected to influence this phenomenon by competing for the fixation site. Poisoning by fluorooacetate can indeed be prevented or reversed by such substances in intact animals and in some isolated systems. Unfortunately, a different interpretation can be placed upon this observation. There is no way of knowing with certainty whether added acetate competes with fluorooacetate or simply bypasses a blockade. To clarify this it is necessary

to marshal the data concerning the various possible blockades which have been tested.

Although it would be extremely helpful to have more data on other compounds, nearly all the detailed investigations concerning inhibition of metabolic processes by the fluorocarboxids have been performed with fluorooacetic or fluorooxyacetic acid. The specificity (or even the existence) of the inhibition of the oxidation of acetate and pyruvate by fluorooacetate has been the chief subject of study, the results of which have been vastly complicated by differences in species or organs employed. (One searches the literature in vain for

mention of the existence of fluoropyruvic acid, $\text{F}-\text{CH}_2-\text{C}(\text{O})-\text{COOH}$, which

ought to be utilized to settle some of these questions.)

It is fairly certain that fluorooacetic acid does not inhibit acetylation of foreign natural amines by mammals. The formation of acetoacetate from acetate is inhibited and in general—*Litomosoides carinii* being a clear exception—the oxidation of pyruvate to acetate may be directly affected while the disposition of the acetate so formed is certainly affected. Acetate probably exerts an inhibitory effect upon pyruvate oxidation after a sufficient amount of it has accumulated. There appears to be no way at this time of choosing between findings which suggest inhibition of fumarate oxidation and those which do not. In this connection it is possible to argue obliquely that, because fluorooacetate and other potent competitive inhibitors of succinic dehydrogenase are relatively stable animal poisons, inhibition of other neighboring steps in the Krebs' cycle is likely to be the cause of death in mammals poisoned with the usual minute doses of fluorooacetate. The effects on citrate and glucose metabolism may best be explained as the indirect result of inhibition at other points.

It is well established that the breakdown of glucose to triose phosphate can occur anaerobically and aerobically, and is a source of much energy. Pyruvate

has long been known as the main end-product of this metabolic chain, lactate

being a simple conversion product of pyruvate. However, in the last decade the fragment, a much discussed entity having many but not all of the characteristics of acetate, has been definitely added as a stage of carbohydrate oxidation

before presenting the schema depicted in Figure 1, it is necessary to bring out

far less well known observations which relate to the possibility of its general

metabolizability and to the action of fluorooacetate. Differences among species with regard to the action of fluorooacetate must be more than a matter of vagary. If

the importance of these systems and pathways to the economy of the organism

is studied, some interesting relations are obtained. Thus, although there are

qualitative differences between Wistar and Sprague-Dawley strains of rats

the way they handle the acetate in their acetate pool, Wistar rats turn over

acetate (12 to 15 mM/100 gram/24 hr.) than do Sprague-Dawley rats (20

to 25 mM/100 gram/24 hr.).

It is possible that some organisms may be very sensitive if odd-numbered

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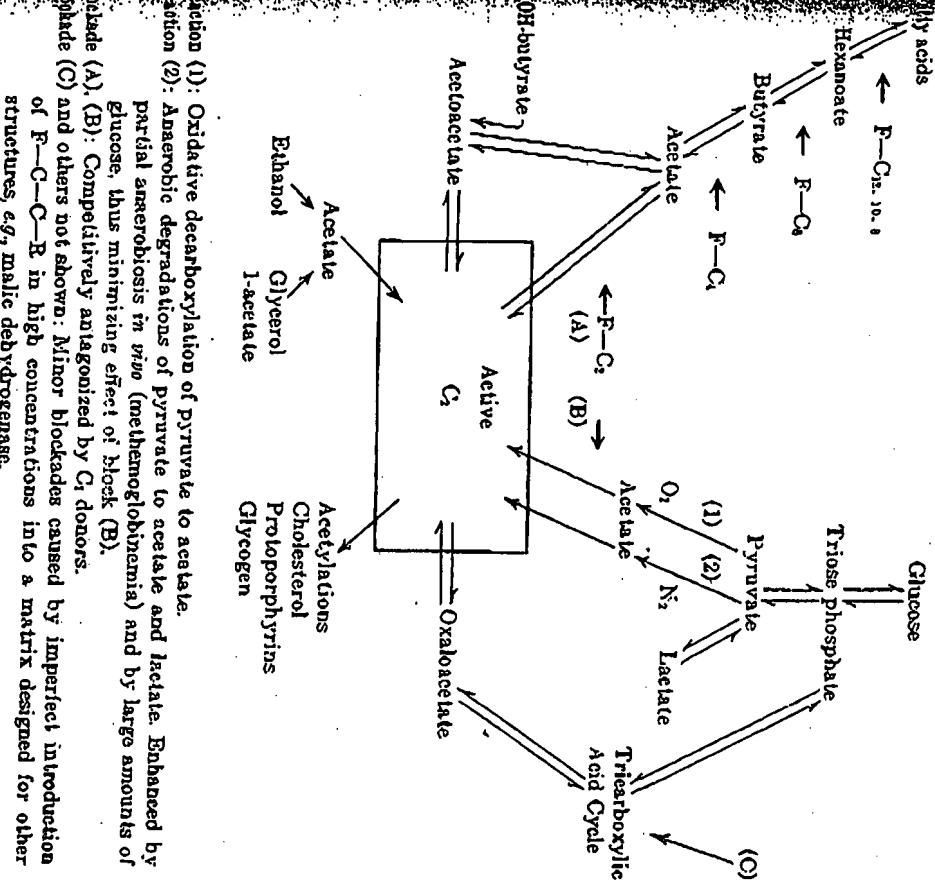
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to 25 mM/100 gram/24 hr.). In addition Wistar rats are unable to convert pyruvate to acetate although Sprague-Dawley rats do so freely (4). The difference in response to fluoracetate is clear; the strain to which acetate is more important is more sensitive to fluoracetate (although more resistant to pyridoxine deficiency (27) or alloxan (82)). It has recently been shown that intact rabbits metabolize formate actively, but that dogs and man are nearly unable to do so incidentally, this accounts for the relative toxicity of methanol to these two latter species (88, 89, 90). Dog muscle, on the other hand, can oxidize β -hydroxybutyrate and acetacetate whereas rabbit muscle can only use acetacetate (124). The differences between dogs and rabbits in response to fluoracetate are striking. Perhaps a similar relation exists when still other species are considered for isolated mammals of the goat, a fluoracetate-sensitive animal, utilize acetate whereas those of the relatively resistant rat and still more resistant mouse do not appear to do so (51). Mention has been made of the ability of guinea pig brain to oxidize added acetate and the relative inability of rabbit brain to do so, the fluoracetate-induced inhibition of acetate oxidation being greater in brain tissue from the guinea pig than from the rabbit. The convulsive pattern of fluoracetate poisoning in the guinea pig and the effects on the heart of the rabbit, which oxidizes acetate vigorously (10, 11, 33), intimate that this is no mere coincidence.

When one views these scattered observations on the relation between fluoracetate action and metabolic pathways in the light of the protective effect of acetate and its donors, it is not unreasonable to suspect that the degree of sensitivity of an animal or its organs to fluoracetate is an indication of a certain characteristic of its acetate metabolism. To establish this suspicion as a fact will require much effort and it is probable that the problem is even more complicated than appears at first. Reference has been made to studies with isolated frog muscle, rabbit intestine and intact animals which show that there are fluoracetate-insensitive metabolic pathways giving rise to sufficient energy to maintain function. Evidence has been adduced to indicate that the breakdown of glucose is not sensitive to fluoracetate and that some such system can be enhanced in the presence of an excess of glucose and insulin. It is highly probable that in addition to individual peculiarities of the metabolic systems for handling C₁-C₄ molecules in a given species a second factor, the glycolytic rate, is important in controlling the sensitivity of the organ or organism. Few comparative data have been published although Wu and Chang (134) have recently pointed out that the glycolytic rate of isolated eel, toad, turtle and rat hearts decreased (in that order) with the resistance to anoxia. Grossly, the sensitivity to fluoracetate likewise decreased in the direction of greater glycolytic activity. It is also well known that very young mammals are very much more resistant to anoxia than are older ones. Nine 24-hour old dogs recently tested in this laboratory were markedly resistant to fluoracetate. Further, the relative sensitivity of various vertebrates to tissue anoxia induced by potassium cyanide (67) is strikingly similar to their ordinary sensitivity to fluoracetate. Farah (50) has called attention to the similarity

FIG. 1. Tentative Localization of Fluoroacid Blockades



between the sensitivities to fluoracetate, cyanide and anoxia of various portions

of the gut, which follow Alvarez's concept of a metabolic gradient. Although it is surprisingly difficult to find accurate comparative data (64), such adult mammals have been found very resistant to anoxia. (21) are also resistant to fluoracetate.

Figure 1, the anaerobic degradation of pyruvate to acetate. (1): Oxidative decarboxylation of pyruvate to acetate. (2): Anaerobic degradations of pyruvate to acetate and lactate. Enhanced by partial anaerobiosis *in vivo* (methemoglobinemia) and by large amounts of glucose, thus minimizing effect of block (B).

(3): (A): (B): Compellatively antagonized by C₁ donors. (C): and others not shown: Minor blockades caused by imperfect introduction of F-C-C-E in high concentrations into a matrix designed for other structures, e.g., malic dehydrogenase.

in Figure 1, the anaerobic degradation of pyruvate to acetate by coupled oxidation-reduction reactions ("dismutation") to lactate and acetate, or by partial anaerobiosis *in vivo* (methemoglobinemia) and by large amounts of glucose, thus minimizing effect of block (B).

(3): (A): (B): Compellatively antagonized by C₁ donors. (C): and others not shown: Minor blockades caused by imperfect introduction of F-C-C-E in high concentrations into a matrix designed for other structures, e.g., malic dehydrogenase.

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phenomenon, however, is outstandingly unexplained. If blockades by the fluorooacids actually occur as indicated, why are these acids so much more toxic than the corresponding carboxylic acids? Perhaps this will become clear as the understanding of fatty acid metabolism increases. On the whole, it seems possible to explain the pharmacological action of Fluoroacetate itself, together with some of the specific variations noted, in terms of the magnitude and character of the acetate and glycolytic metabolism. The characteristic response of an organism or tissue to fluorooacids may be determined by the relative importance of these two pathways.

SUMMARY

Conversion of a metabolic intermediate into a very highly toxic compound by the introduction of a single fluorine atom in a strategic position in the molecule has been described for a number of compounds. It appears to be a useful method for producing agents with which metabolic pathways can be differentiated. A large number of species with a minimum of effort, for it is evident that the agents act by virtue of their close resemblance to natural metabolites. As an example, the variation among species in response to monofluoroacetic acid has been related to certain definite differences in metabolism in the species studied.

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ADDENDUM

The gracious permission of the British Ministry of Supply to refer to information in certain of their classified reports was received too late to permit inclusion of this information in the body of the review. Mention should be made, however, of the high resistance of the *Cercopithecus*, or "green" monkey to methyl fluorooacetate. The LD₅₀ appears to lie above 50 mgm./kgm. according to K. J. C. Pearter and B. A. Kilby (1944). These workers also found that although fluorooacetate is as active as fluorooacetate in intact animals, it is without action on the isolated perfused heart, presumably because this organ can not convert fluorooacetate to fluorooacetic acid.

Definite histologic abnormalities in the myocardium, but no unequivocal changes in the central nervous system, were reported by A. M. Barrett (1944) in guinea pigs and rabbits poisoned repeatedly with methyl fluorooacetate. The beating of heart muscle cells in culture is rapidly inhibited by methyl fluorooacetate but the growth of the cell masses is not affected, according to C. B. Allard and H. B. Fell (1944). The action is specific, for methyl chloroacetate in equivalent concentrations merely kills the cells.

The view that fluorooacetic acid is metabolized to fluorooxic acid, accounting for inhibition of citrate oxidation and the subsequent accumulation of fluorooxyacid, has been advanced by Martius (Ann. d. Chem. 661: 227-232, 1949). This would seem referable to Blockade (C) of Figure 1.

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THE EFFECTS OF DRUGS UPON THE ELECTRICAL ACTIVITY OF THE BRAIN*

JAMES E. P. TOMAN AND JEAN P. DAVIS

Departments of Physiology and Pharmacology, University of Utah College of Medicine, Salt Lake City, Utah

I. INTRODUCTION

It is now twenty years since Hans Berger (20) presented to the world a revolutionary new method for the investigation of the function of the brain in health and disease, namely, *electroencephalography*. His contribution was threefold. To the clinician he gave a diagnostic instrument with sharp differential and localizing value, without which the notable advances of the last two decades in the pharmacological and surgical treatment of central nervous disorders would have been sorely handicapped. To the neurophysiologist he gave a research tool of high precision and relative simplicity for the unraveling of the central nervous system by the monitoring of their electrical signs. Lastly, to all those who strive to understand the mode of operation of the organ of thought, he gave a new point of departure. The brain could no longer be considered a passive switchboard through which impulses coursed on their way to and from the periphery; it was now revealed as a dynamic participant in the affairs of the body, possessing an inherent spontaneous and rhythmic activity which could modify the soma and be modified in turn by the soma.

Berger did not neglect to investigate the effects of drugs upon the electrical activity of the brain (21). He looked for electroencephalographic signs of the central nervous actions of barbital, morphine, cocaine, amyl nitrite, scopolamine, chloroform and other substances then in common clinical use. Thus he opened a field of research which has many potentialities not only for the determination of the mechanism of action of centrally acting drugs, but also for the analysis of the nature of the electroencephalogram itself, for the reason that substances with known specific action are among the most precise tools available for investigations in the biological sciences.

Unfortunately, the rapidly increasing volume of literature in the field of electroencephalography contains relatively few systematic studies of the effects of drugs on the electrical activity of the brain, although there are many empirical reports on the alterations produced by particular chemical agents. Among previous reviews relevant to this subject should be mentioned those of Hayslip (176), Gibbs and Gibbs (135), Fluesinger and Brazier (108), Gibbs (130, 131, 132), Lennox and Lennox (217), and Walter and Walter (331).

The present review will consider primarily the effects of drugs upon the normal human EEG. This will necessarily exclude from discussion most of the large

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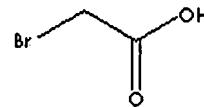
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BROMOACETIC ACID

PRODUCT IDENTIFICATION

CAS NO. 79-08-3
EINECS NO. 201-175-8
FORMULA BrCH₂COOH
MOL WT. 138.95
H.S. CODE



TOXICITY Oral rat LD50: 50 mg/kg

SYNOMYS 2-Bromo acetic acid; alpha-Bromoacetic acid;
Monobromoacetic acid;

DERIVATION**CLASSIFICATION****PHYSICAL AND CHEMICAL PROPERTIES**

PHYSICAL STATE white to light yellow crystalline powder

MELTING POINT 49 - 51 C

BOILING POINT 206 - 208 C

SPECIFIC GRAVITY 1.934

SOLUBILITY IN WATER soluble

pH

VAPOR DENSITY

AUTOIGNITION

NFPA RATINGS

Health: 3; Flammability: 1; Reactivity: 0

REFRACTIVE INDEX

FLASH POINT

STABILITY Stable at ordinary conditions. Hydroscopic.

APPLICATIONS

Bromoacetic Acid is used as a chemical intermediate for the manufacturing other compounds and pharmaceuticals.

SALES SPECIFICATION

APPEARANCE white to light yellow crystalline powder

PURITY 98.0% min

TRANSPORTATION**PACKING**

HAZARD CLASS 3 (Packing Group: II)

UN NO. 1938

OTHER INFORMATION

Hazard Symbols: T C, Risk Phrases: 23/24/25-36, Safety Phrases: 36/37/39-45



CHLOROACETIC ACID

PRODUCT IDENTIFICATION

CAS NO. 79-11-8
EINECS NO. 201-178-4
FORMULA CH_2ClCOOH
MOL WT. 94.50

H.S. CODE**TOXICITY**

SYNOMYS Chloroethanoic acid; Monochloroethanoic acid; ácido cloroacético (Spanish); Acide chloroacétique (French); alpha-Chloroacetic acid; MCA; Monochloroacetic Acid; Monochloroazijnzuur; Monochloressigsäure (German); Acidomonochloroacético (Italian);

DERIVATION acetic acid, chlorine

CLASSIFICATION**PHYSICAL AND CHEMICAL PROPERTIES**

PHYSICAL STATE white solid
MELTING POINT 61 - 62 C
BOILING POINT 188 C (solid), 143 C (80% aqueous solution)
SPECIFIC GRAVITY 1.40 (solid), (1.20 80% aqueous solution)
SOLUBILITY IN WATER Soluble
SOLVENT SOLUBILITY Soluble in ether, chloroform, benzene, and alcohol
pH <1 (80% aqueous solution)

VAPOR DENSITY**AUTOIGNITION**

NFPA RATINGS Health: 3; Flammability: 1; Reactivity: 0

REFRACTIVE INDEX

FLASH POINT 126 C (solid), (1.20 80% aqueous solution)
STABILITY Stable under ordinary conditions. Hygroscopic.

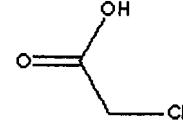
DESCRIPTION AND APPLICATIONS

Monochloroacetic acid, chlorinated simplest carboxylic acid, has electron-withdrawing atom (alpha-chlorine) on the next carbon to acid. Alpha-chlorine makes monochloroacetic acid more acidic than acetic acid. It is used as one of the first choice chemical intermediates for the production of;

- Carboxy Methyl Cellulose and Starch
- Phenoxyacetic Acid
- Thioglycolic Acid
- Cyanoacetic Acid / Malonates / Barbituric Acid
- Caffeine, Betaine, Vitamin B and pharmaceuticals
- Glycine
- Surfactants
- Indigo dyes
- Herbicides

SALES SPECIFICATION

APPEARANCE white flakes
ASSAY 99.0% min



THE MERCK INDEX

AN ENCYCLOPEDIA OF
CHEMICALS, DRUGS, AND BIOLOGICALS

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Bromoacetic acid

p-Bromoaniline

1425

Mon-

USE: In water disinfection; bleaching fibers and silk; manuf medicinal bromine compds, dyestuffs.

1416. **Bromine Pentafluoride.** BrF_5 ; mol wt 174.90. Br 45.69%, F 54.31%. Prepd by fluorination of bromine at 200° in iron or Monel metal apparatus: Ruff, Menzel, *Z. Anorg. Allgem. Chem.* 202, 49 (1931); Kwasnik in *Handbook of Preparative Inorganic Chemistry*, Vol. 1, G. Brauer, Ed. (Academic Press, New York, 2nd ed., 1963) pp 158-159; from F_2 and KBr : Hyde, Boudakian, *Inorg. Chem.* 7, 2648 (1968). Reviews: Kemmitt, Sharp, *Advan. Fluorine Chem.* 4, 243-244 (1965); Stein, "Physical and Chemical Properties of Halogen Fluorides" in *Halogen Chemistry*, Vol. 1, V. Gutmann, Ed. (Academic Press, New York, 1967) pp 133-224; Meinert, *Z. Chem.* 7, 41-57 (1967).

Liquid. Fumes in air. mp -60.5°. bp 40.76°. d_{4}^{25} 2.4604. Trouton constant 23.7. Thermostable up to 460°. Does not attack quartz when dry. *Produces an explosion on contact with water.* Very reactive, usually with conflagration.

Caution: Corrosive and irritating to eyes, skin, mucous membranes.

1417. **Bromine Trifluoride.** BrF_3 ; mol wt 136.90. Br 58.37%, F 41.63%. Prepd by fluorination of bromine at 80°: Lebeau, *Compt. Rend.* 141, 1018 (1905); Prideaux, *J. Chem. Soc.* 89, 316 (1906); Ruff, Braida, *Z. Anorg. Allgem. Chem.* 206, 59 (1932); 214, 91 (1933); Simons, *Inorg. Syn.* 3, 184 (1950); Kwasnik in *Handbook of Preparative Inorganic Chemistry*, Vol. 1, G. Brauer, Ed. (Academic Press, 2nd ed., 1963) pp 156-157. Reviews: Kemmitt, Sharp, *Advan. Fluorine Chem.* 4, 244-245 (1965); Stein, "Physical and Chemical Properties of Halogen Fluorides" in *Halogen Chemistry*, Vol. 1, V. Gutmann, Ed. (Academic Press, New York, 1967) pp 133-224; Meinert, *Z. Chem.* 7, 41-57 (1967).

Colorless liquid; also reported to be pale yellow. Long prisms when solid. mp 8.77°. bp 125.75°. d_{4}^{25} 2.8030. *Smokes in air. Attacks skin. Very reactive.*

Caution: Corrosive and irritating to skin, eyes, mucous membranes, respiratory tract.

USE: Solvent for fluorides.

1418. **Bromoisovalum.** *N*-(*Aminocarbonyl*)-2-bromo-3-ethylbutanamide; (α -bromoisovaleryl)urea; 2-monobromo- α -valerylurea; α -bromo- β -dimethylpropanoylurea; bromoaceton; B.V.U.; Bromural; Bromisoval; Uvaleral; Bromulan; Somnurol; Brovalurea; Dormigene; Isobromyl; Alluval; Ivadorm. $\text{C}_6\text{H}_11\text{BrN}_2\text{O}_2$; mol wt 223.07. C 32.31%, H 9.77%, Br 35.82%, N 12.56%, O 14.34%. $(\text{CH}_3)_2\text{CHCHBrCONH}_2$. Prepn from urea and α -bromoisovaleryl bromide: U.S. pat. 914,518 (1909). Toxicity: R. I. Mrongovius et al., *Clin. Exp. Pharmacol. Physiol.* 3, 443 (1976). Practically tasteless needles, mp 147-149°. Sublimes. lightly sol in cold water, but freely sol in hot water. easily sol in alcohol, ether and in alkaline solns. LD_{50} in ale mice (mmoles/kg): 3.25 i.p. (Mrongovius).

Caution: This substance may be habit forming and is listed in the U.S. Code of Federal Regulations, Title 21 Part 19.1 (1995).

THERAP CAT: Sedative, hypnotic.

1419. **N**-Bromoacetamide. Acetobromamide. $\text{C}_2\text{H}_4\text{BrN}$; mol wt 137.96. C 17.41%, H 2.92%, Br 57.92%, N 1.5%, O 11.60%. CH_3CONHBr . Prepd by treating a cold (0-5°) soln of acetamide dissolved in bromine with ice and aq 50% KOH, allowing to stand for 2-3 hr at 0-5°, then with NaCl, and extracting with chloroform: Oli-Gerold, *Org. Syn.* 31, 17 (1951).

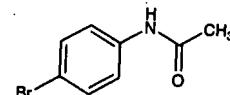
Needles from chloroform + hexane, mp 102-105°. Sol in rm water, freely sol in cold ether. Unstable to light and air.

Monohydrate, rectangular plates from hydr ether, mp 70-70.5°. Freely sol in cold water, alcohol, ether, less freely in chloroform.

Caution: Brominating agent; in oxidation of primary and secondary alcohols.

1420. **p**-Bromoacetanilide. *N*-(*4*-Bromophenyl)acetide; *4*'-bromoacetanilide; monobromoacetanilide; bromoacetanilide; Antiseptic; Asepsin; Bromoantifebrin. $\text{C}_9\text{H}_8\text{BrNO}$; wt 214.06. C 44.89%, H 3.77%, Br 37.33%, N 6.54%, O 7.346 (1874); Merker, Vona, *J. Chem. Ed.* 26, 613

(1949); Kn-santo).



Crystals, from 95% alc, mp 168° (with previous softening). d 1.72. Practically insol in cold water. Sparingly sol in hot water; sol in benzene, chloroform, ethyl acetate; moderately sol in alcohol.

THERAP CAT: Analgesic, antipyretic.

1421. **Bromoacetic Acid.** $\text{C}_2\text{H}_2\text{BrO}_2$; mol wt 138.95. C 17.29%, H 2.18%, Br 57.51%, O 23.03%. BrCH_2COOH . Prepn by bromination of acetic acid: Perkin, Duppa, *Ann.* 108, 106 (1858); from chloroacetic acid and HBr: Lake, Asadorian; Asadorian, Burk, U.S. pats. 2,553,518; 3,130,-222 (1951, 1964, both to Dow); from glycolic acid and HBr: Johnston, U.S. pat. 2,876,255 (1959 to Ethyl Corp.).

Hygroscopic crystals, mp 50°. bp 208°. d 1.93. Very sol in water, alcohol. *Protect from air and moisture.*

Caution: Irritant and corrosive to skin, mucous membranes.

1422. **Bromoacetone.** *1*-Bromo-2-propanone. $\text{C}_3\text{H}_5\text{BrO}$; mol wt 136.98. C 26.31%, H 3.68%, Br 58.33%, O 11.68%. $\text{CH}_3\text{COCH}_2\text{Br}$. Prepn by bromination of acetone: Emmerling, Wagner, *Ann.* 204, 29 (1880); Catch et al., *J. Chem. Soc.* 1948, 272; Ross, U.S. pat. 2,452,154 (1948 to Colgate-Palmolive-Peet); by bromination acetone enol acetate: Magerlein, U.S. pat. 2,752,341 (1956 to Upjohn).

Liquid, mp -36.5°. bp 137°. bp_{50} 63.5-64°. d_{4}^{25} 1.634. n_{D}^{25} 1.4697. Turns violet rapidly even in absence of air. Sparingly sol in water; sol in alcohol, acetone. *Keep tightly closed and protected from light.*

Caution: Violent lacrimator.

USE: Chemical war gas.

1423. **p**-Bromoacetophenone. *1*-(4-Bromophenyl)ethanone; methyl *p*-bromophenyl ketone. $\text{C}_8\text{H}_7\text{BrO}$; mol wt 199.05. C 48.27%, H 3.54%, Br 40.14%, O 8.04%. $\text{BrC}_6\text{H}_4\text{COCH}_3$. Prepd from bromobenzene and acetic anhydride in CS_2 in the presence of anhyd AlCl_3 : Adams, Noller, *Org. Syn.* 5, 17 (1925).

Leaflets from alc, mp 54°. bp_{76} 255.5°; bp_{15} 130°; bp 117°. Easily volatile with steam. Sol in alcohol, ether, glacial acetic acid, benzene, petr ether, carbon disulfide.

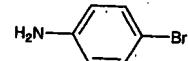
Caution: Oxime, $\text{C}_8\text{H}_8\text{BrNO}$, needles from dil alc, mp 128.5°.

1424. **ω -Bromoacetophenone.** *2*-Bromo-1-phenylethethane; phenacyl bromide. $\text{C}_8\text{H}_7\text{BrO}$; mol wt 199.05. C 48.27%, H 3.54%, Br 40.14%, O 8.04%. $\text{C}_6\text{H}_5\text{COCH}_2\text{Br}$. Prepn from acetophenone and bromine: Rother, Reid, *J. Am. Chem. Soc.* 41, 77 (1919); Cowper, Davidson, *Org. Syn.* 19, 24 (1939); Shevchuk, Dombrovskii, *Zh. Obshch. Khim.* 33(4), 1135 (1963).

Crystals, mp 50°. d 1.65. bp_{20} 133-135°. Irritating vapors. *Lacrimator!* Practically insol in water. Freely sol in alcohol, benzene, chloroform, ether.

Caution: Highly irritating to skin, eyes, mucous membranes.

1425. **p**-Bromoaniline. *4*-Bromobenzeneamine; 4-bromoaniline. $\text{C}_6\text{H}_5\text{BrN}$; mol wt 172.02. C 41.89%, H 3.52%, Br 46.45%, N 8.14%. Prepd by steam distilling sodium hydroxide and *p*-bromoacetanilide: Scott, *J. Chem. Soc.* 123, 3199 (1923); by direct bromination of aniline: Kosolapoff, *J. Am. Chem. Soc.* 75, 3596 (1953).



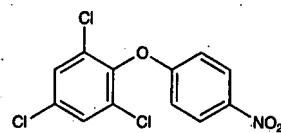
Rhombic crystals from dil alc; mp 66-66.5°. d_{4}^{25} 1.4970 (liq). Very sol in alcohol and ether. Insol in cold water. pK_b (25°): 10.28 also reported as 9.98.

USE: In prep of azo dyes; condensed with formaldehyde in prep of dihydroquinazolines.

Fifth Annual Report on Carcinogens (NTP 89-239, 1989) p 340.

THERAP CAT: Antineoplastic.

2158. Chlornitrofen. *1,3,5-Trichloro-2-(4-nitrophenoxy)benzene; 4-nitrophenyl-2,4,6-trichlorophenyl ether; 2,4,6-trichlorophenyl-4'-nitrophenyl ether; CNP; MO-338; MO; Showrone.* $C_{12}H_6Cl_3NO_3$; mol wt 318.54. C 45.25%, H 1.90%, Cl 33.39%, N 4.40%, O 15.07%. Prepd, not claimed: Neth. pat. Appl. 6,405,727; I. Takayuki *et al.*, U.S. pat. 3,316,080 (1964, 1967 both to Mitsui Chemical); and field studies: G. W. Mason, E. G. Brooker, *Proc. 21st N. Z. Weed Pest Control Conf.* 163 (1968). Properties: J. Kanazawa, *Pestic. Sci.* 12, 417 (1981). Absorption and metabolism in crops: H. Shimotori, S. Kuwatsuka, *J. Pestic. Sci.* 3, 267 (1978). Degradation studies: M. Oyamada, S. Kuwatsuka, *ibid.* 4, 157 (1979). GC determin in fish: T. Yamagishi *et al.*, *Bull. Environ. Contam. Toxicol.* 23, 57 (1979); in river water: H. Kobayashi *et al.*, *J. Chromatog.* 643, 197 (1993).



Pale yellow needles from ethanol, mp 105-106°. Solv: 0.25 mg/l water. Solv at 20-25°: 764 μ g/l water. Vapor pressure (109°): 4.67×10^4 mPa. Partition coefficient (octanol/water): 4709. LC_{50} in carp (48 hr): > 10,000 μ g/l; LC_{50} in flea (3 hr): > 40,000 μ g/l (Kanazawa).

USE: Herbicide.

2159. Chloroacetaldehyde. 2-Chloro-1-ethanal; monochloroacetaldehyde. C_2H_3ClO ; mol wt 78.50. C 30.60%, H 3.85%, Cl 45.16%, O 20.38%. $CICH_2CHO$. Prepd industrially by carefully controlled chlorination of acetaldehyde: Söll, Ger. pat. 844,595 (1943 to Knapsack-Griesheim); Shawinigan Chem. Ltd., Brit. pat. 644,914 (1947); Guinot, Tabuteau, *Compt. Rend.* 231, 234 (1950); Fr. pat. 1,012,991 (1950 to Comp. des Prod. Chim. et Electrometallurg.).

Liquid. Acrid, penetrating odor. bp_{760} 85-86°. The anhydrous substance polymerizes on standing but reverts to the monomer on distillation.

Hemihydrate, probably $CICH_2CH(OH)OCH(OH)CH_2Cl$, platelets from water, mp 43-50°, bp 85.5° with decompr into water and chloraldehyde. Sol in water, alcohol, ether.

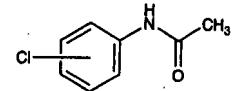
Caution: Potential symptoms of overexposure are irritation of eyes, skin and mucous membranes; skin burns; eye damage; pulmonary edema; skin, respiratory system sensitization; narcosis, coma. See NIOSH Pocket Guide to Chemical Hazards (DHHS/NIOSH 90-117, 1990) p 64.

USE: In the manufacture of 2-aminothiazole; to facilitate bark removal from tree trunks.

2160. Chloroacetamide. 2-Chloroacetamide. C_2H_4ClNO ; mol wt 93.51. C 25.69%, H 4.31%, Cl 37.91%, N 14.98%, O 17.11%. $CICH_2CONH_2$. Prepn from ethyl chloroacetate and ammonia: Jacobs, Heidelberger, *Org. Syn. Coll. vol. I*, 153 (2nd ed., 1941); from chloroacetyl chloride and ammonium acetate: Finan, Fothergill, *J. Chem. Soc.* 1962, 2824.

Crystals from water, mp 119-120°, bp ~225° (dec). Sol in 10 parts water, 10 parts abs alc; very slightly sol in ether. Two isomorphous crystalline modifications: α -form, "stable form", obtained by sublimation and by crystallization from nonpolar solvents; β -form, "unstable form", obtained by quenching the melt or by crystallization from polar solvents, see Katayama, *Acta Crystallogr.* 9, 986 (1956); 10, 468 (1957); B. Kalyanaraman *et al.*, *J. Cryst. Mol. Struct.* 8, 175 (1978).

2161. Chloroacetanilide. C_8H_8ClNO ; mol wt 169.61. C 56.65%, H 4.75%, Cl 20.90%, N 8.26%, O 9.43%. Prepn of *m*-, *o*- and *p*-isomers: Roberts *et al.*, *J. Org. Chem.* 24, 654 (1959). Sepn of *o*- and *p*-isomers: Orton, Bradford, *J. Chem. Soc.* 1927, 986.



m-Chloroacetanilide, N-(3-chlorophenyl)acetamide; chloroacetanilide. Needles from 50% glacial acetic acid 77-78°. Readily sol in alcohol, benzene, carbon dis very slightly sol in ligroin. uv max (95% ethanol): 207 (log ε 4.19).

o-Chloroacetanilide. Needles from dil glacial acetic mp 87-88°. Sublimes at about 50-60°. Practically ins water, alkalis; sol in alc; more sol in benzene than sponding *p*-isomer. uv max (95% ethanol): 240 nm (log ε 4.02).

p-Chloroacetanilide. Orthorhombic crystals from glacial acetic acid, alc, or acetone. mp 178-179°. Practically insol in water; readily sol in alc, ether, ch disulfide; slightly sol in CCl_4 , benzene. uv max (95% ethanol): 249 nm (log ε 4.25).

2162. Chloroacetic Acid. Chloroethanoic acid; chloroacetic acid; MCA. $C_2H_3ClO_2$; mol wt 94.50. 25.42%, H 3.20%, Cl 37.52%, O 33.86%. $CICH_2COOH$. Made by chlorination of glacial acetic acid in presence small amount of sulfur or iodine; also by hydrolysis chlorethylene with 90% H_2SO_4 . Lab prep: Gatter Wieland, *Praxis des organischen Chemikers* (de Gruyter, Berlin, 40th ed., 1961) pp 109, 110. Manuf: Faith, Clark's *Industrial Chemicals*, F. A. Lowenheim, M. Moran, Eds. (Wiley-Interscience, New York, 4th ed., pp 254-257). Toxicity: Woodward *et al.*, *J. Ind. Hyg. col.* 23, 78 (1941).

Colorless or white, deliquescent crystals. d 1.580. α , β and γ forms having mp 63°, 55-56° and 50° respect mp for acid of commerce is 61-63°. bp for all forms. Very sol in water; sol in alcohol, benzene, chloroform, *Keep well closed and in a cool place.*

Sodium salt, $C_2H_3ClNaO_2$, *Monoxone*. White cry solid. Solv in water at 20°: 85 g/100 ml. LD_{50} in mice, guinea pigs (mg/kg): 76, 255, 80 orally (Woodward).

Caution: Irritating to skin, mucous membranes. USE: Herbicide. Manuf various dyes and other chemicals.

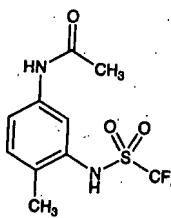
2163. Chloroacetic Anhydride. *Chloroacetic anhydride; monochloroacetic acid anhydride; sym-dichloroacetic anhydride.* $C_2H_4Cl_2O_2$; mol wt 170.98. C 28.10%, Cl 41.47%, O 28.07%. ($CICH_2CO$)₂. Prepn: treating monochloroacetic acid in tetrahydrofuran methoxyacetylene at 15° and distilling the mixture: Epton *et al.*, *J. Chem. Soc.* 1954, 1860; by mixing monochloroacetic acid and HCN with 1,4-dioxane satd with HCl; fractionating the mixture: Krieble, Smellie, *U.S. pat. 390,106* (1945); by heating chloroacetyl chloride with monochloroacetic acid in the presence of $AlCl_3$; Stross Schwegler, *U.S. pat. 1,713,104* (1929 to Dow).

Prisms from benzene; d_{4}^{20} 1.5494; mp 46°. bp_{760} 163°; bp_{62} 149°; bp_{42} 126°; bp_{10} 109-110°; bp_{40} 118°. Freely sol in ether, chloroform; slightly sol in benzene, tically insol in cold ligroin.

USE: In *N*-acetylation of amino acids in alkaline prepn of cellulose chloroacetates.

2164. Chloroacetone. 1-Chloro-2-propanone; chloroacetone; monochloroacetone; monochloracetone; acetone chloride; chloropropanone; 1-chloro-2-ketopropane; 1-chloropropane. C_3H_6ClO ; mol wt 92.52. C 38.94%, H 6.44%, Cl 38.32%, O 17.29%. $CICH_2COCH_3$. Prepn by the of chlorine upon diketene: A. B. Boese, Jr., *U.S. pat. 2,209,683* (1940 to Carbide and Carbon Chem.); by nati on of acetone: E. J. Rahrs, *U.S. pat. 2,235,451* (1941 to Eastman Kodak); G. H. Morey, *U.S. pat. 2,229,651* (1941 to Commercial Solvents Corp.). Stabilization: U.S. pat. 2,229,651 (1941 to Commercial Solvents Corp.). E. J. Rahrs, *U.S. pat. 2,263,010* (1941 to Eastman Kodak). Forms binary azeotropes with many organic liquids. Horsley, *Azeotropic Data, Advances in Chemistry Series* 6 (Washington, 1952) p 73. Reacts with aryl

40.54%, H 3.74%, F 19.24%, N 9.46%, O 16.20%, S 10.82%.
Prepn: Harrington *et al.*, U.S. pat. 3,639,474 (1972 to Minnesota Mining & Mfg.).



mp 175-176.5°.

USE: Plant growth retardant.

4199. Fluorine, F; at. wt 18.9984032; at. no. 9; valence 1; elemental state F₂. A halogen. Occurrence in earth's crust 0.065% by wt. Natural abundance of isotopes: ¹⁹F 100%; ¹⁸F ($T_{1/2}$ 109.7 min) is prepared in nuclear reactors. Does not occur in elemental state in nature. Most important sources are fluorite, cryolite, and fluorapatite: Finger, "Fluorine Resources and Fluorine Utilization" in *Advances in Fluorine Chemistry*, vol. 2, M. Stacey *et al.*, Eds. (Butterworths, London, 1961) pp 35-54. Discovered in 1771 by Scheele. Isolated in 1886 by electrolyzing a soln of potassium fluoride in anhyd. hydrogen fluoride at -23°, using platinum-iridium electrodes: Moissan, *Compt. Rend.* 102, 1543 (1886); 103, 202, 256. Subsequent methods of prep: Ruff, *Ber.* 69, 181 (1936); Henne, *J. Am. Chem. Soc.* 60, 96 (1938). ¹⁸F has been used to study fluorine exchange reactions: Dave, Sowerby, "Isotopic Halogen Exchange Reactions" in *Halogen Chemistry*, vol. 1, V. Gutmann, Ed. (Academic Press, New York, 1967) pp 41-132. Toxicity data: Keplinger, Suissa, *Am. Ind. Hyg. Assoc. J.* 29, 10 (1968). Reviews: A. J. Rudge, *The Manufacture and Use of Fluorine and its Compounds* (Oxford University Press, 1962); O'Donnell, "Fluorine" in *Comprehensive Inorganic Chemistry*, vol. 2, J. C. Bailar, Jr. *et al.*, Eds. (Pergamon Press, Oxford, 1973) pp 1009-1106; A. J. Woytek in Kirk-Othmer *Encyclopedia of Chemical Technology*, vol. 10 (Wiley-Interscience, New York, 3rd ed., 1980) pp 630-654. Pale yellow, diatomic gas. mp -219.61° (53.54°K); bp -188.13° (85.02°K); d (liq. -188.13°) 1.5127; vapor pressure data: Hu *et al.*, *J. Am. Chem. Soc.* 75, 5642 (1953); White *et al.*, *ibid.* 76, 2584 (1954). Crit temp: -129°; crit pressure: 55 atm. Most reactive nonmetal; higher oxidation potential than ozone; most electronegative element; E° (calc) $\frac{1}{2}F/F^-$ 2.9 V. F-F bond weaker than Cl-Cl and Br-Br bonds; enthalpy of dissociation: 37.7 kcal. Reacts vigorously with most oxidizable substances at room temp, frequently with ignition. Combines directly or indirectly, to form fluorides with all the elements except helium, neon and argon. Dec water, giving hydrofluoric acid, HF, oxygen fluoride, OF₂, hydrogen peroxide, oxygen and ozone. Reacts with nitric acid, forming the explosive gas, fluorine nitrate, NO₂F; with sulfuric acid, giving fluorosulfuric acid, HFSO₃. Yields the metal fluorides, water, oxygen and oxygen fluoride when made to react with metal hydroxides in the cold. Reacts violently with organic compds, usually with disintegration of the molecule. Under controlled conditions, however, hydrocarbon vapors may be fluorinated with elemental fluorine. Solid fluorine explodes when brought in contact with liquid hydrogen. Under ordinary conditions it does not react directly with oxygen, nor does it react with oxides of sodium, potassium or calcium. LC₅₀ (1 hr) inhalation by rats, mice, guinea pigs: 185, 150, 170 ppm (by vol) (Keplinger, Suissa).

Caution: Potential symptoms of overexposure are irritation of eyes, nose and respiratory tract; laryngeal spasms, bronchial spasms; pulmonary edema; eye and skin burns. See *NIOSH Pocket Guide to Chemical Hazards* (DHHS/NIOSH 90-117, 1990) p 116. Chronic ingestion of high concentrations from water supply can cause mottled enamel of teeth and osteosclerosis. See *Clinical Toxicology of Commercial Products*, R. E. Gosselin *et al.*, Eds. (Williams & Wilkins, Baltimore, 5th ed., 1984) Section III, pp 185-193.

Fluorine

4200. Fluorine Dioxide, Dioxygen difluoride; mol wt 70.00. F 54.28%, O 45.72%. Prepd from Ruff, Menzel, *Z. Anorg. Allgem. Chem.* 211, 217, 85 (1934); Goetschel *et al.*, *J. Am. Chem. Soc.* 81, 2233 (1952). Toxicity data: Darmer *et al.*, *Am. Assoc. J. 33*, 661 (1972). Review: Kemmitt, Sharp, *Advan. Fluorine Chem.* 4, 213-314 (1965).

Thermally unstable gas at room temp; begins to decompose at -100°. Pale yellow gas; red liq at low temp. mp -154°C (119°K). Described as brown gas, red liq and orange solid. -163.5°, probably contained other fluorine-containing compounds as impurities: Goetschel *et al.*, loc. cit. Dizing and fluorinating agent.

4201. Fluorine Monoxide, Oxygen fluoride; F₂O; mol wt 54.00. F 70.37%, O 29.63%. Prepd by passing fluorine slowly through an aq NaOH soln: Inorg. Syn. 1, 109 (1939); Schnizlein *et al.*, *J. Phys. Chem.* 56, 233 (1952). Toxicity data: Darmer *et al.*, *Am. Assoc. J. 33*, 661 (1972). Review: Kemmitt, Sharp, *Fluorine Chem.* 4, 213-314 (1965).

Colorless gas. Yellowish-brown when liq. Peculiar. Does not attack glass in the cold. Corrodes metal. Reacts very slowly with water. The gas may be dissolved in water unchanged for a month. d (liq. -224°) 1.23-1.25. bp -145.3°. Trouton constant 20.63. water (0°) 6.8 ml gas/100 ml H₂O. LC₅₀ (1 hr) inhalation by rats, mice: 2.6, 1.5 ppm (Darmer).

Caution: Potential symptoms of overexposure are headache; respiratory system irritation, pulmonary edema; eye and skin burns. See *NIOSH Pocket Guide to Chemical Hazards* (DHHS/NIOSH 90-117, 1990) p 116.

4202. Fluorine Nitrate, Nitroxy fluoride; nitrofluoride; nitryl hypofluorite. FNO₂; mol wt 81.01. 23.45%, N 17.29%, O 59.25%. FNO₂. Prepd by the action of fluorine on nitric acid: Cady, *J. Am. Chem. Soc.* 2635 (1934); Ruff, Kwasnik, *Angew. Chem.* 48, 218 (1935); Kwasnik in *Handbook of Preparative Inorganic Chemistry*, vol. 1, G. Brauer, Ed. (Academic Press, New York, 1963) pp 187-189. Reviews: Kemmitt, Sharp, *Advan. Fluorine Chem.* 4, 216-218 (1965); Woolf, *ibid.* 5, 1-30 (1966); Schmutzler, *Angew. Chem. Int. Ed.* 7, 440-455 (1968).

Colorless gas. Moldy, acrid odor. mp -175°. -45.9°. d (liq at bp) 1.507. d²-193.2 (solid) 1.951. Trouton const 20.8. The liq explodes on slight percussion. Sol in water to OF₂, O₂, HF and HNO₃. Sol in alcohol. Conflagrates on contact with alcohol, ether, aniline. May be stored in vacuum-sealed glass ampuls cooled by liquid nitrogen. Powerful oxidizing agent.

USE: Oxidizing agent in rocket propellants.

4203. Fluorine Perchlorate, Chlorine tetroxyfluoride, ClFO₄; mol wt 118.45. Cl 29.93%, F 16.04%, O 54.03%. Prepd by passing fluorine over cold 72% perchloric acid in platinum apparatus: Rohrback, Cady, *J. Am. Chem. Soc.* 69, 677 (1947).

Colorless gas. Pungent, acrid odor. mp -167.5°. -15.9°. Exploses on the slightest provocation, i.e., on contact with rough surfaces, dust, grease, rubber, on melting, distilling, etc.

Caution: Attacks lungs even in traces.

4204. Fluoroacetamide, Fluoroacetic acid amide; monofluoroacetamide; 1081; Fluorakil 100; Fussol, CH₃FNO; mol wt 77.06. C 31.17%, H 5.23%, F 24.65%, 18.18%, O 20.76%. CH₃FCONH₂. Numerous synths. e.g. from fluoroacetyl chloride and NH₃; Truce, *J. Am. Chem. Soc.* 70, 2828 (1948). Mode of action study and toxicity data: F. Matsumura, R. D. O'Brien, *Biochem. Pharmacol.* 12, 1201 (1963).

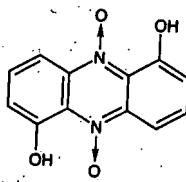
Crystals. Sublimes on heating. Freely sol in water, sol in acetone; sparingly sol in chloroform. LD₅₀ i.p. in mice 11 mg/kg (Matsumura, O'Brien).

USE: Rodenticide. Insecticide proposed mainly for use on fruits to combat scale insects, aphids, and mites.

4205. Fluoroacetic Acid, Fluoroethanoic acid; gitter poison. C₂H₃FO₂; mol wt 78.04. C 30.78%, H 3.87%, 24.34%, O 41.00%. CH₂FCOOH. Occurs in "Gibberella

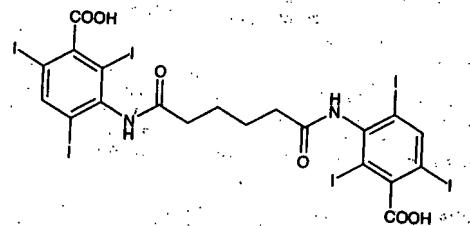
USE: Chlorinating and oxidizing agent.
THERAP CAT: Anti-infective (topical).

5042. Iodinin. *1,6-Phenazinediol 5,10-dioxide*; 1,5-dihydroxyphenazine *N,N*-dioxide; $C_{12}H_8N_2O_4$; mol wt 244.21. C 59.02%, H 3.30%, N 11.47%; O 26.21%. Antibiotic pigment from *Chromobacterium Iodinum*; Clemo, McIlwain, *J. Chem. Soc.* 1938, 479; Hegedüs in *Emil Borell Jubilee Volume* (1946), p 388; from *Waksmania aerata* and *Pseudomonas iodinum*; Gerber, Lechevalier, *Biochemistry* 3, 598 (1964). Structure: Clemo, Daglish, *J. Chem. Soc.* 1950, 1481. Synthesis: Matsumura, Takeda, *Nippon Kagaku Zasshi* 81, 515 (1960), *C.A.* 56, 470a (1962). Biosynthetic studies on incorporation of shikimic acid, *q.v.*, into iodinin; U. Hollstein *et al.*, *Tetrahedron Letters* 1978, 2987; R. B. Herbert *et al.*, *J. Chem. Soc. Perkin Trans. I* 1979, 2411; T. Etherington *et al.*, *ibid.* 2416.



Purple crystals with coppery sheen from chloroform; dec 236°. pK 12.5. Stable in acid; unstable in alkali. Sol in benzene, toluene, xylene, carbon disulfide, chloroform, ethyl acetate. Slightly sol in hot alc. Practically insol in cold alc, ether, acetic acid, petr ether, amyl alcohol. Insol in water. Sol in coned sulfuric acid and in glacial acetic acid with red color. Sol in NaOH solns giving brilliantly blue solns which deposit green crystals of the unstable sodium deriv.

5043. Iodipamide. *3,3'-(1,6-Dioxo-1,6-hexanediyliimino)bis[2,4,6-triiodobenzoic acid]; 3,3'-(adipoyldiimino)bis[2,4,6-triiodobenzoic acid]; N,N'-adipylbis(3-amino-2,4,6-triiodobenzoic acid); adipic acid di(3-carboxy-2,4,6-triiodobenzoic acid); adipodione; Cholografin; Cholospect; $C_{20}H_{14}I_6N_2O_6$; mol wt 1139.77. C 21.08%, H 1.24%, I 66.81%, N 2.46%, O 8.42%. Prepn: Priebe, Rutkowski, U.S. pat. 2,776,241 (1957 to Schering AG); Kotler-Brajtburg *et al.*, *Roczniki Chem.* 36, 763 (1962), *C.A.* 58, 5568a (1963). Purification: Cassebaum, Drux, East Ger. pat. 33,738 (1964), *C.A.* 63, 11441b (1965). Pharmacology: Fischer, Varga, *Acta Physiol.* 38, 135 (1970). Metabolic studies: Kiyono *et al.*, *Radioisotopes* 20, 78 (1971). Pharmacology and toxicity study: F. J. Rosenberg *et al.*, *Invest. Radiol.* 15, S142 (1980). Comprehensive description: H. H. Lerner in *Analytical Profiles of Drug Substances* vol. 3, K. Flörey, Ed. (Academic Press, New York, 1974) pp. 333-363.*



Crystals, dec 306-308°. n_{D}^{25} 1.3294 (c = 0.445 in methanol). Soly at 20°: methanol 0.8%; ethanol 0.3%; acetone 0.2%; ether 0.1%. Practically insol in water, benzene. LD_{50} in mice, rats (mg/kg): 2380 ± 290, 4430 ± 310 i.v. (Rosenberg).

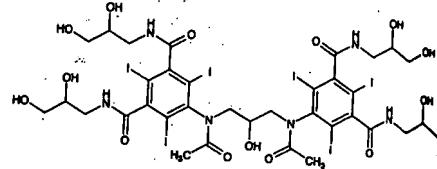
Disodium salt, $C_{20}H_{12}I_6N_2Na_2O_6$. Prepd by dissolving the free acid in a dil aq soln of NaOH and buffering to pH 6.5-7.7. Used as 20% soln.

Bis[N-methylglucamine] salt, $C_{44}H_{48}I_6N_4O_{16}$. *iodipamide meglumine*, *Biligrain*, *Cholegrafin*, *Endocistobil*, *Endografin*, *Intrabilix*, *Transbilix*. More sol in water than the disodium salt. Used as 40% soln.

1000 Acetyl
opaque medium
cystographic).

THERAP CAT (VET): Diagnostic aid (radiopaque medium).

5044. Iodixanol. *5,5'-[2-Hydroxy-1,3-propanediyl]bis[N,N'-bis(2,3-dihydroxypropyl)-2,4,6-triiodobenzoic acid]; 5,5'-(2-hydroxyimino)bis(2,3-dihydroxypropyl)-2,4,6-triiodoisophthalamide; 1,3-bis(acetylaminooxy)-N,N'-bis(2,3-dihydroxypropylaminocarbonyl)-2,4,6-triiodobenzoic acid; 2,5410-3A; Acupaque; $C_{28}H_{44}I_6N_6O_{15}$; mol wt 1550.19. C 27.12%, H 2.14%, I 49.12%, N 5.42%, O 15.48%. Nonionic, dimeric x-ray contrast medium. Prepn: P. E. Hansen *et al.*, *Eur. pat. 108,638* (1984 to Nyegaard), *C.A.* 101, 151598 (1984). HPLC determin in plasma: H. Nomura *et al.*, *J. Chromatogr.* 572, 333 (1991). Clinical pharmacokinetic studies in cerebral arteriography: Y. Palmers *et al.*, *Radiol.* 17, 203 (1993); in cardiac angiography: J. A. H. H. H. H. Conroy *et al.*, *Clin. Radiol.* 49, 337 (1994). Use as gradient medium: T. Ford *et al.*, *Anal. Biochem.* 220, 220 (1994); J. Graham *et al.*, *ibid.* 367.*



mp 240-250°. Soluble in water. Viscosity (37°) 11.1 mgI/ml). 50% aqueous soln (w/v): viscosity 8.1 mgI/ml. LD_{50} i.v. in mice: 21 gI/kg (Palmers).

THERAP CAT: Diagnostic aid (radiopaque medium).

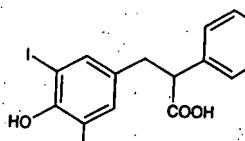
5045. Iodized Oil. Lipiodol. An iodine addition product of vegetable oils, contg 38-42% organically combined iodine.

Thick, viscous, oily liq; alliaceous odor; oleaginous due on exposure to air and light, becoming brown. Keep small, nearly full containers, protected from light.

THERAP CAT: Diagnostic aid (radiopaque medium).

5046. Iodoacetic Acid. C_2H_3IO ; mol wt 185.95. 12.92%, H 1.63%, I 68.25%, O 17.21%. CH_3COOH by treating chloroacetic acid in acetone soln with NaI. Colorless or white crystals. mp 82-83°. Sol in water, very slightly sol in ether.

5047. Iodoaliphionic Acid. *4-Hydroxy-3,5-diodo- α -phenylbenzenepropanoic acid; β -(4-hydroxy-3,5-diodophenyl)- α -phenylpropionic acid; 3,5-diodo- α -phenylbenzoic acid; Coletrast; Priodax; Pheniodol; Dikol; Iodol; Biliognost; Tenicid; Biliselectant. $C_{15}H_{14}I_2O_3$; mol wt 494.07. C 36.47%, H 2.45%, I 51.37%, O 9.71%. Prepd by iodiinating β -(4-hydroxyphenyl)- α -phenylpropionic acid with iodine in an aq soln of alkali iodide in the presence of NH_3 or with iodine in acetic anhydride as the iodiinating agent, cf. Brit. pat. 559,024 (1944), *C.A.* 40, 1883 (1946). Prepn of optically active forms: Tullar, Hoppe, U.S. pat. 2,552,696 (1951 to Sterling Drug). Toxicity data: Hoppe, S. Archer, *Am. J. Roentgenol. Rad. Ther.* 63, 15 (1953).*



Weed Susceptibility Chart

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The herbicide ratings used in this chart are averages for the performance of these herbicides in the state when the herbicide is applied at labeled rates for crop production. Results may vary with location, rate of application, growth stage, and climatic conditions. Individuals are encouraged to alter the ratings to reflect their individual experiences with these herbicides under local conditions.

Ratings: "C" = weed is generally controlled, "P" = weed is injured or partially controlled, "N" = weed is not normally controlled, "-" = not sufficient information to rate the performance of this herbicide on the weed.

WEEDS		HERBICIDES	
COMMON NAME	BOTANICAL NAME ¹	COMMON NAME	TRADE NAME ²
BROADLEAVED WEEDS			
BINDWEED(Pr) ⁴	<i>Convolvulus arvensis</i>	ALACHLOR	= LASSO
BINDWEED(Se) ³	<i>Convolvulus arvensis</i>	ATRAZINE	= AATREX
CARROT, WILD	<i>Daucus carota</i>	BENEFIN	= BALAN
CHICKWEED	<i>Stellaria media</i>	BENSULIDE	= BETASAN, PREFAR, PRESAN
CHINESE THORNAPPLE	<i>Datura ferox</i>	BENTAZON	= BASAGRAN
CLOVER	<i>Medicago and Melilotus spp.</i>	BROMACIL	= HYVAR
COCKLEBUR	<i>Xanthium strumarium</i>	BROMOXYNIL	= BUCTRIL
CUDWEED	<i>Gnaphalium spp.</i>	CARFENTRAZONE	= SHARK
CURLY DOCK(Pr)	<i>Rumex crispus</i>	CLETHODIM	= PRISM, ENVOY
CURLY DOCK(Se)	<i>Rumex crispus</i>	CLOPYRALID	= TRANSLINE, STINGER
DAISY, ENGLISH	<i>Bellis perennis</i>	CHLORSULFURON	= GLEAN, TELAR
DANDELION(Pr)	<i>Taraxacum officinale</i>	CYCLOATE	= RO-NEET
DANDELION(Se)	<i>Taraxacum officinale</i>	DAZOMET	= BASAMID
DODDER	<i>Cuscuta spp.</i>	DCPA	= DACTHAL
FIDDLENECK	<i>Amsinckia menziesii</i>	DICAMBA	= BANVEL, CLARITY, VANQUISH
FILAREE	<i>Erodium spp.</i>	DICHLOBENIL	= CASORON, NOROSAC
FLEABANE, HAIRY	<i>Conyza bonariensis</i>	DICLOFOP	= HOELON
FLUVELIN, SHARPPONT	<i>Kickxia elatine</i>	DIFENZOQUAT	= AVENGE
GOOSEFOOT, NETTLELEAF	<i>Chenopodium murale</i>	DIQUAT	= REWARD
GROUNDCHERRY	<i>Physalis spp.</i>	DIMETHENAMID	= OUTLOOK
GROUNDSEL, COMMON	<i>Senecio vulgaris</i>	DITHIOPYR	= DIMENSION
HENBIT	<i>Lamium amplexicaule</i>	DIURON	= KARMEX, DIREX
HORSETAIL, SCOURING RUSH	<i>Equisetum spp.</i>	DSMA	= DSMA
HORSEWEED, MARETAIL	<i>Conyza canadensis</i>	ENDOTHAL	= AQUATHOL, ENDOTHAL, HYDROTHOL
KNOTWEED	<i>Polygonum spp.</i>	EPTC	= EPTAM, ERADICANE
KOCHIA	<i>Kochia scoparia</i>	ETHAFLURALIN	= CURBIT, SONALAN
KYLLINGA, GREEN	<i>Kyllinga brevifolia</i>	ETHOFUMESATE	= NORTRON, PROGRASS
LAMBSQUARTERS, COMMON	<i>Chenopodium album</i>	FENOXAPROP	= ACCLAIM, PUMA, WHIP
LONDON ROCKET	<i>Sisymbrium irio</i>	FLUAZIFOP	= FUSILADE
MALLOW, CHEESEWEED	<i>Malva spp.</i>	FLUMIOXAZIN	= CHATEAU
MINERS LETTUCE	<i>Claytonia perfoliata</i>	GLUFOSINATE	= FINALE, LIBERTY, RELY
MORNINGGLORY, JAPANESE	<i>Ipomoea nil</i>	GLYPHOSATE	= ROUNDUP ⁵ , TOUCHDOWN ⁶ , RODEO, OTHERS
MUSTARD	<i>Brassica spp.</i>	HALOSULFURON	= MANAGE, PERMIT
NETTLE, BURNING	<i>Urtica urens</i>	HEXAZINONE	= VELPAR, PRONONE
NIGHTSHADE, BLACK	<i>Solanum nigrum</i>	IMAZAMOX	= RAPTOR
NIGHTSHADE, HAIRY	<i>Solanum sarrachoides = S. physalifolium</i>	IMAZAPIC	= PLATEAU
NUTSEDGE, PURPLE	<i>Cyperus rotundus</i>	IMAZAPYR	= ARSENAL, CHOPPER, HABITAT, STALKER
NUTSEDGE, YELLOW	<i>Cyperus esculentus</i>	IMAZETHAPYR	= PURSUIT
ONION, WILD	<i>Allium canadense</i>	ISOXABEN	= GALLERY
PEPPERWEED, PERENNIAL	<i>Lepidium latifolium</i>	LINURON	= LOROX
PIGWEE	<i>Amaranthus spp.</i>	MCPA	= MCPA

<u>WEEDS</u>		<u>HERBICIDES</u>	
COMMON NAME	BOTANICAL NAME ¹	COMMON NAME	TRADE NAME ²
<u>BROADLEAVED WEEDS</u>			
PINEAPPLE WEED	<i>Matricaria matricarioides</i> = <i>Chamomilla suaveolens</i>	MECOPROP	= MCPP
PLANTAIN, BUCKHORN (Pr ⁴)	<i>Plantago lanceolata</i>	METHAM	= VAPAM, SECTAGON
PLANTAIN, BUCKHORN (Se ⁵)	<i>Plantago lanceolata</i>	METOLACHLOR	= DUAL, PENNANT
PRICKLY LETTUCE	<i>Lactuca serriola</i>	METRIBUZIN	= SENCOR
PUNCTUREVINE	<i>Tribulus terrestris</i>	MSMA	= MSMA, BUENO 6
PURSLANE	<i>Portulaca oleracea</i>	NAPROPAMIDE	= DEVRIOL
RADISH, WILD	<i>Raphanus spp.</i>	NICOSULFURON	= ACCENT
REDMAIDS	<i>Calandrinia ciliata</i>	NORFLURAZON	= SOLICAM, ZORIAL, PREDICT
RUSSIAN THISTLE	<i>Salsola tragus</i>	ORYZALIN	= SURFLAN
SHEPHERD'S-PURSE	<i>Capsella bursa-pastoris</i>	OXADIAZON	= RONSTAR
SMARTWEED	<i>Polygonum spp.</i>	OXYFLUORFEN	= GOAL
SOWTHISTLE, ANNUAL	<i>Sonchus oleraceus</i>	PARAQUAT	= GRAMOXONE
SPEEDWELL, PERSIAN	<i>Veronica persica</i>	PENDIMETHALIN	= PROWL, PENDULUM, PRE-M
SPURGE, SPOTTED	<i>Euphorbia maculata</i>	PELARGONIC ACID	= SCYTHE
STARTHISTLE, YELLOW	<i>Centaurea solstitialis</i>	PHENMEDIPHAM	= BETAMIX
SUNFLOWER, COMMON	<i>Helianthus annuus</i>	PRODIAMINE	= BARRICADE, ENDURANCE
SWINECRESS	<i>Coronopus spp.</i>	PROMETRYN	= CAPAROL
TANSY MUSTARD, FLIXWEED	<i>Descurainia spp.</i>	PRONAMIDE	= KERB
THISTLE, CANADA	<i>Cirsium arvense</i>	PYRAZON	= PYRAMIN
TURKEY MULLEIN	<i>Eremocarpus setigerus</i>	PYRITHOBAC	= STAPLE
VELVETLEAF	<i>Abutilon theophrasti</i>	RIMSULFURON	= SHADEOUT, MATRIX
WILLOWHERB, PANICLE	<i>Epilobium brachycarpum</i>	SETHOXYDIM	= POAST
WOODSORREL, CREEPING	<i>Oxalis corniculata</i>	SIMAZINE	= PRINCEP
<u>GRASSES</u>			
BARLEY, HARE	<i>Hordeum murinum</i>	THIAZOPYR	= VISOR
BARNYARDGRASS	<i>Echinochloa crus-galli</i>	TRICLOPYR	= TURFLON, GARLON, REMEDY, GRANDSTAND, RENNOVATE
BERMUDAGRASS(Pr)	<i>Cynodon dactylon</i>	TRIFLURALIN	= TREFLAN, TRILIN
BERMUDAGRASS(Se)	<i>Cynodon dactylon</i>	2,4-D	= 2,4-D
BLUEGRASS, ANNUAL	<i>Poa annua</i>	2,4-DB	= 2,4-DB, BUTYRAC
BLUEGRASS, BULBOUS	<i>Poa bulbosa</i>		
BROME, DOWNY; CHEATGRASS	<i>Bromus tectorum</i>		
BROME, RIPGUT	<i>Bromus rigidus</i>		
CANARYGRASS	<i>Phalaris spp.</i>		
CEREALS	<i>Volunteer cereals</i>		
CRABGRASS	<i>Digitaria spp.</i>		
DALLISGRASS(Pr)	<i>Paspalum dilatatum</i>		
DALLISGRASS(Se)	<i>Paspalum dilatatum</i>		
FALL PANICUM	<i>Panicum dichotomiflorum</i>		
FESCUE	<i>Festuca spp.</i>		
FOXTAIL, YELLOW	<i>Setaria pumila</i>		
GOOSEGRASS	<i>Eleusine indica</i>		
HARE BARLEY	<i>Hordeum leporinum</i>		
JOHNSONGRASS(Pr)	<i>Sorghum halepense</i>		
JOHNSONGRASS(Se)	<i>Sorghum halepense</i>		
OAT, WILD	<i>Avena fatua</i>		
RABBITSFOOT, POLYPOGON	<i>Polypogon monspeliensis</i>		
RESCUEGRASS	<i>Bromus catharticus</i>		
RYEGRASS, ITALIAN	<i>Lolium multiflorum</i>		
SANDBUR	<i>Cenchrus spp.</i>		
SPRANGLETOP, MEXICAN	<i>Leptochloa uninervia</i>		
STINKGRASS	<i>Eragrostis cilianensis</i>		

¹COMMON AND BOTANICAL NAMES FROM: Weed Science Society of America <http://www.wssa.net/weedname.html>

² MOST COMMON NAMES USED, OTHER TRADENAMES POSSIBLE.

³Se = SEEDLING

⁴Pr = PERENNIAL

⁵ GLYPHOSATE ISOPROPYLAMINE SALT

⁶ GLYPHOSATE TRIMETHYLSULFONIUM SALT

Weed Susceptibility Chart

	ALACHLOR	ATRAZINE	BENEFIN	BENSULIDE	BENTAZON	BROMACIL	BROMOXYNIL	CARFENTRAZONE	CLETHODIM	CLOPYRALID
BROADLEAF										
BINDWEED(P)	N	N	N	N	N	P	N	P	N	N
BINDWEED(S)	N	C	P	N	N	C	P	C	N	P
CARROT, WILD	-	-	-	-	-	C	-	-	N	P
CHICKWEED, COMMON	C	C	C	P	N	C	N	P	N	P
CLOVER	N	C	N	N	-	C	N	-	N	C
COCKLEBUR	-	C	N	N	C	C	P	P	N	C
CUDWEED	N	P	N	N	N	C	C	-	N	C
CURLY DOCK(P)	N	N	N	N	N	C	N	-	N	P
CURLY DOCK(S)	C	C	P	N	N	C	C	-	N	C
DAISY, ENGLISH	-	-	-	N	-	-	N	N	N	-
DANDELION(P)	N	N	N	N	N	C	N	-	N	P
DANDELION(S)	N	C	N	N	N	C	C	-	N	C
DODDER	N	N	N	N	N	N	N	P	N	N
FIDDLERNECK	-	C	P	N	-	C	C	C	N	N
FILAREE	-	P	N	N	-	C	N	-	N	P
FLEABANE, HAIRY	N	C	N	N	N	C	C	N	N	-
FLUVELIN	-	N	-	-	-	-	-	-	N	-
GOOSEFOOT, NETTLELEAF	P	C	C	P	N	C	C	-	N	N
GROUNDCHERRY	C	C	N	N	C	C	C	C	N	P
GROUNDSEL, COMMON	N	C	N	N	-	C	C	-	N	C
HENBIT	-	C	N	N	N	C	P	-	N	N
HORSETAIL	N	N	N	N	N	N	N	-	N	N
HORSEWEED, MARESTAIL	N	C	N	N	N	C	C	N	N	P
KNOTWEED	N	C	C	C	-	C	P	-	N	N
KOCHIA	P	C	P	P	P	C	C	-	N	C
KYLLINGA, GREEN	-	-	N	N	-	-	N	-	N	N
LAMBSQUARTERS	P	C	C	C	C	C	C	-	N	N
LONDON ROCKET	N	C	N	N	P	C	C	C	N	P
MALLOW, CHEESEWEED	P	C	N	N	C	C	P	C	N	N
MINERS LETTUCE	-	C	P	N	-	P	P	-	N	N
MORNINGGLORY, JAPANESE	N	C	N	N	P	C	C	C	N	N
MUSTARD	N	C	N	N	C	C	C	P	N	N
NETTLE, BURNING	C	C	N	P	-	C	P	C	N	-
NIGHTSHADE, BLACK	C	C	N	N	P	C	P	C	N	P
NIGHTSHADE, HAIRY	C	C	N	N	C	C	C	C	N	P
NUTSEDGE, PURPLE	N	N	N	N	N	C	N	N	N	N
NUTSEDGE, YELLOW	P	N	N	N	P	C	N	N	N	N
ONION, WILD	-	-	-	N	-	-	N	-	N	N
PEPPERWEED, PERENNIAL	N	N	N	N	N	-	N	N	N	N
PIGWEEED	C	C	C	C	C	C	P	C	N	N
PINEAPPLE WEED	-	C	N	N	N	C	P	N	N	C
PLANTAIN, BUCKHORN(P)	-	P	N	N	N	P	N	-	N	N
PLANTAIN, BUCKHORN(S)	-	C	N	N	N	C	C	-	N	N
PRICKLY LETTUCE	N	C	N	N	P	C	C	-	N	C
PUNCTUREVINE	-	P	P	N	N	C	C	-	N	N
PURSLANE	C	C	C	C	N	C	N	N	N	N
RADISH, WILD	N	C	N	N	P	C	C	P	N	N
REDMAIDS	-	-	-	-	-	-	-	-	-	N
RUSSIAN THISTLE	P	C	P	N	N	C	C	-	N	N
SHEPHERD'S-PURSE	N	C	N	N	-	C	C	P	N	P
SMARTWEED	P	C	P	N	N	C	P	-	N	P
SOWTHISTLE, ANNUAL	P	C	N	N	-	C	C	N	N	P
SPEEDWELL	-	-	-	-	-	-	-	N	-	N
SPURGE, SPOTTED	-	P	N	N	N	P	C	-	N	N
STARTHISTLE, YELLOW	-	C	N	N	-	C	P	N	N	C
SUNFLOWER, COMMON	N	C	N	N	C	C	C	-	N	C
SWINE CRESS	P	-	N	N	-	C	N	-	N	N
TANSY MUSTARD	-	C	N	N	-	C	C	-	N	N
THISTLE, CANADA	N	P	-	N	-	N	N	-	N	C
THORNAPPLE, CHINESE	N	-	N	N	-	C	C	-	N	P
TURKEY MULLEIN	-	N	N	N	N	P	N	-	N	N
VELVETLEAF	P	P	N	N	C	P	C	C	N	N
WILLOWHERB, PANICLE	-	-	N	N	-	C	-	-	N	N
WOODSORREL, CREEPING	N	-	N	N	N	C	N	-	N	N
GRASSES										
BARLEY, HARE	P	C	C	P	N	C	N	N	C	N
BARNYARDGRASS	C	C	C	C	N	C	N	N	C	N
BERMUDAGRASS(P)	N	N	N	N	N	C	N	N	P	N
BERMUDAGRASS(S)	N	C	C	C	N	C	N	N	C	N
BLUEGRASS, ANNUAL	C	C	C	C	N	C	N	N	C	N
BLUEGRASS, BULBOUS	-	-	-	-	-	-	N	N	C	N
BROME, DOWNY	C	C	P	P	N	C	N	N	C	N
BROME, RIPGUT	-	P	P	-	N	C	N	N	C	N
CANARYGRASS	C	C	C	C	N	C	N	N	C	N
CEREALS, VOLUNTEER	N	C	P	N	N	C	N	N	C	N
CRABGRASS	C	P	C	C	N	C	N	N	C	N
DALLISGRASS(P)	N	N	N	N	N	C	N	N	N	N
DALLISGRASS(S)	C	C	C	C	N	C	N	N	P	N
FESCUE	-	-	-	-	N	-	N	N	-	-
FOXTAIL, YELLOW	C	C	C	C	N	C	N	N	C	N
GOOSEGRASS	C	C	P	C	N	C	N	N	C	N
JOHNSONGRASS(P)	N	N	N	N	P	N	N	N	C	N
JOHNSONGRASS(S)	C	C	C	C	N	C	N	N	C	N
OAT, WILD	N	C	P	N	N	C	N	N	C	N
PANICUM, FALL	C	C	C	C	N	C	N	N	-	N
RABBITFOOT, POLYPOGON	-	-	-	-	-	-	N	N	C	N
RESCUEGRASS	-	-	C	N	N	C	N	N	C	N
RYEGRASS, ITALIAN	C	C	C	P	N	C	N	N	C	N
SANDBUR	C	P	P	P	N	C	N	N	C	N
SPRAGLETOP, MEXICAN	C	C	P	C	N	C	N	N	C	N
STINKGRASS	C	C	C	C	N	C	N	N	C	N

Weed Susceptibility Chart

	CHLORSULFURON	CYCLOATE	DACTHAL	DAZOMET	DICAMBA	DICHLOBENIL	DICLOFOP	DIFENZOQUAT	DIQUAT	DIMETHENAMID
BROADLEAF										
BINDWEED(Pr)	-	N	N	P	P	P	N	N	N	N
BINDWEED(Se)	-	N	N	P	P	C	N	N	P	-
CARROT, WILD	-	-	N	-	C	-	N	-	N	-
CHICKWEED, COMMON	C	C	C	C	C	C	N	N	C	C
CLOVER	P	P	N	N	C	P	N	N	P	C
COCKLEBUR	-	N	N	C	C	P	N	N	P	-
CUDWEED	-	P	N	C	P	C	N	N	N	-
CURLY DOCK(Pr)	P	N	N	C	P	N	N	N	N	-
CURLY DOCK(Se)	C	P	C	C	C	C	N	N	C	-
DAISY, ENGLISH	-	-	-	-	-	N	N	-	-	-
DANDELION(Pr)	P	N	N	P	P	N	N	N	N	-
DANDELION(Se)	C	N	C	C	C	C	N	N	N	-
DOODER	-	N	C	N	N	C	N	N	N	-
FIDDLERNECK	C	C	C	C	C	C	N	N	P	-
FILAREE	C	N	P	P	C	P	N	N	P	-
FLEABANE, HAIRY	-	C	N	C	C	P	N	N	C	-
FLUVELLIN	-	N	N	-	-	-	-	-	-	-
GOOSEFOOT, NETTLELEAF	-	C	C	C	C	C	N	N	C	-
GROUNDCHERRY	-	C	C	C	C	C	N	N	C	C
GROUNDSEL, COMMON	C	C	N	C	C	C	N	N	C	-
HENBIT	C	C	P	C	P	C	N	N	C	-
HORSETAIL	C	N	N	-	N	C	N	N	N	-
HORSEWEED, MARESTAIL	C	C	N	C	C	P	N	N	P	-
KNOTWEED	P	P	P	C	C	C	N	N	N	P
KOCHIA	C	P	P	C	C	-	N	N	C	-
KYLLINGA, GREEN	-	-	N	C	N	-	N	-	-	-
LAMBSQUARTERS	C	C	C	C	C	C	N	N	C	C
LONDON ROCKET	C	N	P	C	C	C	N	N	C	-
MALLOW, CHEESEWEED	C	P	P	P	P	C	N	N	N	-
MINERS LETTUCE	C	C	-	C	-	C	-	-	-	-
MORNINGGLORY, JAPANESE		N	N	-	C	C	N	N	P	-
MUSTARD	C	N	P	C	C	C	N	N	C	-
NETTLE, BURNING	-	P	P	C	P	C	N	N	P	-
NIGHTSHADE, BLACK	-	P	P	C	C	C	N	N	C	-
NIGHTSHADE, HAIRY	-	C	P	C	C	C	N	N	C	-
NUTSEDGE, PURPLE	-	P	N	P	N	P	N	N	N	-
NUTSEDGE, YELLOW	-	P	N	P	N	C	N	N	N	-
ONION, WILD	-	-	N	-	C	-	N	-	-	-
PEPPERWEED, PERENNIAL	C	N	N	C	P	N	N	N	N	-
PIGWEE	C	C	C	C	P	N	N	N	C	C
PINEAPPLE WEED	-	C	N	C	C	C	N	N	P	-
PLANTAIN, BUCKHORN(Pr)	-	N	N	C	P	-	N	N	N	-
PLANTAIN, BUCKHORN(Se)	-	N	N	C	C	C	N	N	C	-
PRICKLY LETTUCE	C	C	N	C	C	C	N	N	P	-
PUNCTUREVINE	C	P	P	C	C	C	N	N	C	-
PURSLANE	-	C	C	C	C	C	N	N	C	C
RADISH, WILD	C	N	N	C	C	C	N	N	C	-
REDMAIDS	-	C	-	C	-	C	-	-	-	-
RUSSIAN THISTLE	C	P	C	C	C	C	N	N	C	-
SHEPHERD'S-PURSE	C	P	N	C	C	C	N	N	P	-
SMARTWEED	-	P	C	C	C	-	N	N	-	-
SOWTHISTLE, ANNUAL	C	C	P	C	C	C	N	N	P	-
SPEEDWELL	P	C	-	-	-	-	-	-	-	-
SPURGE, SPOTTED	-	C	P	C	P	C	N	N	C	-
STARTHISTLE, YELLOW	P	-	N	-	C	C	N	N	C	-
SUNFLOWER, COMMON	C	N	P	C	C	C	N	N	P	-
SWINE CRESS	C	-	N	C	P	-	N	N	P	-
TANSY MUSTARD	C	N	P	C	C	-	N	N	C	-
THISTLE, CANADA	P	-	N	P	P	-	N	-	-	N
THORNAPPLE, CHINESE	-	-	N	-	C	-	N	N	P	-
TURKEY MULLEIN	P	N	N	C	P	-	N	N	-	-
VELVETLEAF	-	N	N	-	C	-	N	N	P	N
WILLOWHERB, PANICLE	-	-	N	C	-	-	N	N	P	-
WOODSORREL, CREEPING	-	-	N	P	P	C	N	N	P	-
GRASSES										
BARLEY, HARE	N	C	C	C	N	C	N	N	P	C
BARNYARDGRASS	N	C	C	C	N	P	P	N	P	C
BERMUDAGRASS(Pr)	N	N	N	P	N	P	N	N	P	-
BERMUDAGRASS(Se)	-	C	C	C	N	P	N	N	P	-
BLUEGRASS, ANNUAL	-	C	C	C	N	C	-	N	P	C
BLUEGRASS, BULBOUS	N	-	-	-	N	-	-	-	-	-
BROME, DOWNY	N	C	C	C	N	C	C	N	P	-
BROME, RIPGUT	N	C	C	C	N	C	-	N	-	-
CANARYGRASS	N	C	C	C	N	C	P	N	P	-
CEREALS, VOLUNTEER	N	N	P	C	N	C	N	N	P	-
CRABGRASS	-	C	C	C	N	P	C	N	C	C
DALLISGRASS(Pr)	N	N	N	P	N	N	N	N	N	-
DALLISGRASS(Se)	-	C	C	C	P	C	N	N	N	C
FESCUE	P	C	-	-	N	-	-	-	-	-
FOXTAIL, YELLOW	P	C	C	C	N	C	C	N	C	C
GOOSEGRASS	-	C	C	C	N	C	N	N	P	-
JOHNSONGRASS(Pr)	N	N	N	P	N	N	N	N	N	-
JOHNSONGRASS(Se)	-	C	C	C	N	N	N	N	C	-
OAT, WILD	N	C	P	C	N	C	C	C	P	C
PANICUM, FALL	-	C	C	C	N	-	-	N	-	-
RABBITFOOT, POLYPOGON	N	-	-	-	N	-	-	-	P	-
RESCUEGRASS	-	C	N	C	N	-	-	N	-	-
RYEGRASS, ITALIAN	N	C	C	C	N	C	C	N	P	C
SANDBUR	-	C	C	C	N	C	C	N	P	-
SPRAGLETOP, MEXICAN	-	C	C	C	N	C	-	N	N	-
STINKGRASS	-	C	C	C	N	P	-	-	P	-

Se = SEEDLING, "P" = PERENNIAL, "—" = NO RATINGS, "N" = NO CONTROL, "P" = PARTIAL CONTROL, "C" = CONTROL

Weed Susceptibility Chart

	DITHIOPYR	DIURON	DSMA	ENDOTHAL	EPTC	ETHALFLURALIN	ETHOFUMESATE	FENOKAPROP	FLUAZIFOP	FLUMIOXAZIN
BROADLEAF										
BINDWEED(Pt)	N	N	N	N	N	-	N	N	N	N
BINDWEED(Se)	P	C	N	N	N	-	N	N	N	-
CARROT, WILD	-	P	-	-	N	-	-	N	N	-
CHICKWEED, COMMON	C	C	P	P	C	C	C	N	N	C
CLOVER	N	P	N	N	N	P	C	N	N	C
COCKLEBUR	-	C	P	P	N	-	P	N	N	-
CUDWEED	P	C	N	N	P	P	C	N	N	-
CURLY DOCK(Pt)	N	P	N	N	N	-	N	N	N	-
CURLY DOCK(Se)	-	C	N	C	C	C	-	N	N	-
DAISY, ENGLISH	N	N	-	-	-	-	-	N	N	N
DANDELION(Pt)	N	N	N	N	N	-	N	N	N	-
DANDELION(Se)	P	C	N	C	C	-	-	N	N	-
DODDER	C	N	N	N	N	-	N	-	N	-
FIDDLERNECK	C	C	N	C	C	C	C	N	N	C
FILAREE	P	C	N	N	P	-	P	N	P	C
FLEABANE, HAIRY	N	P	N	P	C	-	P	N	N	C
FLUVELIN	-	-	-	-	-	-	-	-	N	-
GOOSEFOOT, NETTLELEAF	C	C	N	N	C	-	C	N	N	C
GROUNDCHERRY	C	C	N	P	C	-	C	N	N	C
GROUNDSEL, COMMON	P	N	N	N	C	C	P	N	N	C
HENBIT	C	C	C	P	C	P	P	N	N	C
HORSETAIL	N	N	N	N	N	-	N	N	N	N
HORSEWEED, MARESTAIL	N	P	N	N	C	-	P	N	N	C
KNOTWEED	C	C	N	C	P	P	C	N	N	C
KOCHIA	-	P	-	-	P	-	P	N	N	-
KYLINGA, GREEN	N	-	P	N	P	N	-	N	N	N
LAMBSQUARTERS	P	C	N	P	C	-	C	N	N	C
LONDON ROCKET	C	C	N	N	C	N	N	N	N	-
MALLOW, CHEESEWEED	P	P	N	N	N	P	P	N	N	C
MINERS LETTUCE	C	C	-	-	N	C	-	-	N	C
MORNINGGLORY, JAPANESE	-	C	P	-	-	-	C	N	N	C
MUSTARD	C	C	N	P	N	P	P	N	N	C
NETTLE, BURNING	C	C	N	C	C	P	P	N	N	C
NIGHTSHADE, BLACK	C	C	N	N	P	P	P	N	N	C
NIGHTSHADE, HAIRY	C	C	N	P	C	P	C	N	N	C
NUTSEDGE, PURPLE	N	N	P	N	P	-	P	N	N	N
NUTSEDGE, YELLOW	N	N	P	N	P	-	P	N	N	P
ONION, WILD	-	-	-	-	N	-	-	N	N	-
PEPPERWEED, PERENNIAL	N	N	N	N	N	-	N	N	N	N
PIGWEED	P	C	N	N	C	C	P	N	N	C
PINEAPPLE WEED	C	C	N	-	C	P	P	N	N	C
PLANTAIN, BUCKHORN(Pt)	N	N	N	N	-	-	N	N	N	-
PLANTAIN, BUCKHORN(Se)	-	C	N	-	-	-	N	N	N	-
PRICKLY LETTUCE	-	C	N	P	C	C	C	N	N	C
PUNCTUREVINE	-	P	P	N	N	N	C	N	N	-
PURSLANE	C	C	N	N	C	C	C	N	N	C
RADISH, WILD	-	C	N	N	N	-	N	N	N	-
REDMAIDS	C	-	-	-	C	-	-	N	N	C
RUSSIAN THISTLE	-	P	N	N	P	-	P	N	N	-
SHEPHERD'S-PURSE	C	C	N	P	P	P	N	N	N	C
SMARTWEED	-	C	-	P	C	-	N	N	N	-
SOWTHISTLE, ANNUAL	P	C	N	P	C	C	C	N	N	C
SPEEDWELL	-	-	-	-	C	-	-	N	N	-
SPURGE, SPOTTED	C	N	N	N	N	-	N	N	N	-
STARTHISTLE, YELLOW	N	C	-	-	-	-	N	N	N	C
SUNFLOWER, COMMON	N	C	N	N	P	-	N	N	N	-
SWINE CRESS	-	C	-	N	N	-	N	N	N	-
TANSY MUSTARD	-	C	N	P	N	-	-	N	N	-
THISTLE, CANADA	N	N	-	-	N	-	-	N	N	-
THORNAPPLE, CHINESE	N	-	N	-	N	-	N	N	N	-
TURKEY MULLEIN	-	N	N	N	N	-	-	N	N	-
VELVETLEAF	C	-	-	-	-	-	-	N	N	-
WILLOWHERB, PANICLE	P	N	N	-	-	-	-	N	N	C
WOODSORREL, CREEPING	C	C	P	N	-	-	N	N	N	-
GRASSES										
BARLEY, HARE	C	C	N	N	C	C	C	-	C	-
BARNYARDGRASS	C	C	P	N	C	C	P	C	C	-
BERMUDAGRASS(Pt)	N	N	N	N	N	-	N	P	C	-
BERMUDAGRASS(Se)	C	N	N	N	C	-	-	P	C	-
BLUEGRASS, ANNUAL	C	C	N	C	C	C	C	N	N	-
BLUEGRASS, BULBOUS	-	N	-	-	-	-	-	-	-	-
BROME, DOWNY	C	C	N	N	C	C	-	-	C	-
BROME, RIPGUT	C	C	-	N	C	C	C	-	C	-
CANARYGRASS	C	C	N	P	C	C	C	C	C	-
CEREALS, VOLUNTEER	P	C	N	P	C	C	C	N	C	-
CRABGRASS	C	C	C	N	C	-	P	C	C	P
DALLISGRASS(Pt)	N	N	C	N	N	-	N	N	N	-
DALLISGRASS(Se)	C	C	C	N	C	-	-	N	-	-
FESCUE	C	-	-	-	C	-	-	-	-	-
FOXTAIL, YELLOW	C	C	-	N	C	-	C	C	C	-
GOOSEGRASS	P	C	C	N	C	C	N	P	C	-
JOHNSONGRASS(Pt)	N	N	N	N	N	-	N	N	C	-
JOHNSONGRASS(Se)	C	C	C	N	C	-	C	C	C	-
OAT, WILD	P	P	N	N	C	C	C	C	C	C
PANICUM, FALL	-	N	C	N	C	-	-	-	-	-
RABBITFOOT, POLYPOGON	-	C	-	-	C	-	-	C	C	-
RESCUEGRASS	C	-	-	N	C	C	N	-	-	-
RYEGRASS, ITALIAN	C	C	N	N	C	N	N	N	C	-
SANDBUR	C	C	N	N	C	-	-	-	C	-
SPRANGLETOP, MEXICAN	C	N	N	N	C	-	-	N	P	-
STINKGRASS	C	C	-	N	C	-	C	-	C	-

Ss = SEEDLING, "P" = PERENNIAL, "-" = NO RATINGS, "N" = NO CONTROL, "P" = PARTIAL CONTROL, "C" = CONTROL

Weed Susceptibility Chart

	GLUFOSINATE	GLYPHOSATE	HALOSULFURON	HEXAZINONE	IMAZAMOX	IMAZAPIC	IMAZAPYR	IMAZETHAPYR	ISOXABEN
BROADLEAF									
BINDWEED(Pr)	P	P	N	N	P	-	C	N	C
BINDWEED(Se)	C	C	-	C	P	-	-	-	C
CARROT, WILD	-	C	-	-	-	-	-	-	-
CHICKWEED, COMMON	C	C	C	C	C	-	C	P	C
CLOVER	C	P	-	C	N	N	P	N	C
COCKLEBUR	C	C	C	C	P	N	C	P	-
CUDWEED	C	C	-	C	-	N	C	P	C
CURLY DOCK(Pr)	-	P	-	N	N	-	C	P	N
CURLY DOCK(Se)	C	C	-	C	N	-	-	P	C
DAISY, ENGLISH	-	C	-	-	-	N	-	-	-
DANDELION(Pr)	-	C	-	C	P	N	-	P	C
DANDELION(Se)	C	C	-	C	P	N	C	P	C
DODDER	P	C	-	N	P	-	-	P	C
FIDDLENECK	C	C	-	C	P	-	C	P	C
FLAREE	C	P	-	P	C	C	C	P	C
FLEABANE, HAIRY	C	C	-	C	-	N	C	N	C
FLUVELLIN	-	P	-	-	-	-	-	-	-
GOOSEFOOT, NETTLELEAF	C	C	-	C	-	-	C	P	C
GROUNDCHERRY	C	C	-	C	-	-	-	C	C
GROUNDSEL, COMMON	C	C	C	C	-	N	-	P	C
HENBIT	C	C	-	P	P	-	C	P	C
HORSETAIL	N	P	N	N	-	-	-	N	N
HORSEWEED, MARESTAIL	C	C	-	C	-	N	C	N	C
KNOTWEED	P	P	-	C	P	-	C	C	C
KOCHIA	C	C	P	C	P	-	C	P	-
KYLINGA, GREEN	-	N	P	-	-	-	-	-	N
LAMBSQUARTERS	C	C	N	C	C	-	C	P	C
LONDON ROCKET	C	C	C	C	P	C	C	C	C
MALLOW, CHEESEWEED	C	P	C	P	C	-	P	C	C
MINERS LETTUCE	-	C	-	C	C	-	-	C	C
MORNINGGLORY, JAPANESE	C	P	P	C	P	-	C	P	C
MUSTARD	C	C	C	P	C	C	-	C	C
NETTLE, BURNING	C	N	C	C	P	-	C	C	C
NIGHTSHADE, BLACK	C	C	N	C	C	-	-	C	C
NIGHTSHADE, HAIRY	C	C	N	C	C	-	-	C	C
NUTSEDGE, PURPLE	P	P	C	N	-	-	-	P	N
NUTSEDGE, YELLOW	P	P	C	N	-	-	-	P	N
ONION, WILD	-	-	-	-	-	-	-	-	-
PEPPERWEED, PERENNIAL	-	P	N	-	P	C	C	P	N
PIGWEEED	C	C	P	C	C	-	C	C	C
PINEAPPLE WEED	C	C	-	P	-	N	-	-	-
PLANTAIN, BUCKHORN(Pr)	-	P	-	P	N	-	-	-	N
PLANTAIN, BUCKHORN(Se)	C	C	-	C	P	-	C	-	-
PRICKLY LETTUCE	C	C	C	C	N	N	C	N	C
PUNCTUREVINE	C	C	-	C	P	-	C	C	C
PURSLANE	C	P	P	C	P	-	C	C	C
RADISH, WILD	C	C	C	C	C	C	C	P	C
REDMAIDS	-	C	-	-	-	-	-	-	-
RUSSIAN THISTLE	C	C	-	C	P	-	C	P	C
SHEPHERD'S-PURSE	C	C	C	C	C	C	C	C	C
SMARTWEED	C	C	P	-	P	-	C	C	-
SWTHISTLE, ANNUAL	C	C	C	C	P	N	P	N	C
SPEEDWELL	-	C	-	-	-	-	-	-	-
SPURGE, SPOTTED	C	C	-	C	P	-	C	P	C
STARTHISTLE, YELLOW	C	C	-	C	N	N	C	P	-
SUNFLOWER, COMMON	C	C	C	C	C	N	C	P	C
SWINE CRESS	-	C	N	C	C	C	C	C	-
TANSY MUSTARD	C	C	-	C	C	C	C	C	-
THISTLE, CANADA	-	P	N	N	P	N	-	-	N
THORNAPPLE, CHINESE	-	C	-	-	C	-	-	-	-
TURKEY MULLEIN	C	P	-	P	-	-	-	-	-
VELVETLEAF	-	P	C	-	C	-	C	C	C
WILLOWHERB, PANICLE	C	P	-	-	-	-	C	C	P
WOODSORREL, CREEPING	-	C	-	P	-	-	C	-	C
GRASSES									
BARLEY, HARE	-	C	-	P	C	C	-	-	N
BARNYARDGRASS	C	C	P	C	P	-	C	P	N
BERMUDAGRASS(Pr)	P	C	N	N	-	-	-	N	N
BERMUDAGRASS(Se)	C	C	N	P	-	-	P	P	N
BLUEGRASS, ANNUAL	C	C	N	P	P	C	C	N	N
BLUEGRASS, BULBOUS	-	C	-	-	C	C	C	-	-
BROME, DOWNY	C	C	-	C	C	C	C	N	N
BROME, RIPGUT	C	C	-	P	C	C	C	N	N
CANARYGRASS	C	C	-	C	P	C	C	P	N
CEREALS, VOLUNTEER	C	C	-	P	C	C	-	P	N
CRABGRASS	C	C	C	C	P	-	C	P	N
DALLISGRASS(Pr)	P	C	N	P	-	-	-	N	N
DALLISGRASS(Se)	C	C	N	C	-	-	P	-	N
FESCUE	-	C	-	-	P	-	-	-	-
FOXTAIL, YELLOW	C	C	-	C	C	-	C	C	N
GOOSEGRASS	-	C	-	-	-	-	C	-	N
JOHNSONGRASS(Pr)	P	C	-	N	N	-	-	P	N
JOHNSONGRASS(Se)	C	C	-	C	P	-	C	C	N
OAT, WILD	C	C	-	P	C	P	C	P	N
PANICUM, FALL	-	C	-	C	-	-	C	P	N
RABBITFOOT, POLYPOGON	-	C	-	-	-	-	-	-	-
RESCUEGRASS	-	C	-	P	C	C	-	N	N
RYEGRASS, ITALIAN	-	C	N	C	C	C	C	P	N
SANDBUR	C	C	-	C	C	-	C	P	N
SPRAGLETOP, MEXICAN	C	C	-	-	-	-	P	N	N
STINKGRASS	C	C	-	-	-	-	C	-	N

Se = SEEDLING, "P" = PERENNIAL, "-" = NO RATINGS, "N" = NO CONTROL, "P" = PARTIAL CONTROL, "C" = CONTROL

Weed Susceptibility Chart

	LINURON	MCPA	MECOPROP	METHAM	METOLACHLOR	METRIBUZIN	MSMA	NAPROPAIMIDE	NICOSULFURON	NORFLURAZON
BROADLEAF										
BINDWEED(Pt)	N	N	N	P	N	N	N	N	-	N
BINDWEED(Se)	N	N	N	P	N	N	N	N	-	P
CARROT, WILD	N	-	-	-	N	-	-	-	-	-
CHICKWEED, COMMON	C	P	P	C	C	C	P	C	-	P
CLOVER	P	N	P	N	N	N	N	P	-	N
COCKLEBUR	C	C	C	C	-	P	P	C	N	C
CUDWEED	P	P	P	C	N	-	N	P	-	P
CURLY DOCK(Pt)	N	P	P	C	N	N	N	N	-	N
CURLY DOCK(Se)	C	C	C	C	C	C	N	P	-	N
DAISY, ENGLISH	-	-	-	-	-	-	-	-	-	-
DANDELION(Pt)	N	N	N	P	N	N	N	N	-	N
DANDELION(Se)	N	N	C	C	N	C	N	C	-	N
DOODER	N	N	N	N	N	N	N	N	-	N
FIDOLENECK	C	P	P	C	N	C	N	C	-	P
FILAREE	C	P	-	C	N	C	N	C	-	P
FLEABANE, HAIRY	N	C	C	C	N	C	N	N	-	P
FLUVELIN	-	-	-	-	-	-	-	-	-	-
GOOSEFOOT, NETTLELEAF	C	C	C	C	P	C	N	C	-	C
GROUNDCHERRY	C	C	C	C	C	P	N	N	-	C
GROUNDSEL, COMMON	C	N	N	C	N	P	N	C	-	P
HENBIT	C	N	N	C	-	C	C	N	-	P
HORSETAIL	N	N	N	-	N	N	N	N	-	N
HORSEWEED, MARESTAIL	N	C	C	C	N	P	N	N	-	P
KNOTWEED	P	P	P	C	N	-	N	C	-	P
KOCHIA	C	-	-	C	P	C	-	C	P	-
KYLINGA, GREEN	N	-	-	-	N	-	P	N	-	P
LAMBSQUARTERS	C	C	C	C	P	C	N	C	-	P
LONDON ROCKET	C	C	C	C	N	C	N	C	-	P
MALLOW, CHEESEWEED	C	P	P	N	P	C	N	P	-	P
MINERS LETTUCE	-	-	-	C	-	-	-	-	-	P
MORNINGGLORY, JAPANESE	P	P	P	P	N	N	P	P	P	C
MUSTARD	C	C	C	C	N	C	N	P	-	P
NETTLE, BURNING	C	P	C	C	C	C	N	P	-	C
NIGHTSHADE, BLACK	C	C	C	C	C	P	N	N	N	C
NIGHTSHADE, HAIRY	P	C	C	C	N	N	N	N	-	C
NUTSEDGE, PURPLE	N	N	N	P	N	N	P	N	-	P
NUTSEDGE, YELLOW	P	N	N	C	P	P	C	N	N	P
ONION, WILD	-	-	-	-	-	-	-	-	-	-
PEPPERWEED, PERENNIAL	N	N	N	C	N	N	N	N	-	N
PIGWEE	C	C	C	C	C	N	C	C	C	P
PINEAPPLE WEED	C	P	N	C	-	P	N	P	-	P
PLANTAIN, BUCKHORN(Pt)	-	P	P	C	N	N	N	N	-	N
PLANTAIN, BUCKHORN(Se)	-	C	C	C	-	N	N	N	-	-
PRICKLY LETTUCE	C	C	C	C	N	C	N	C	-	P
PUNCTUREVINE	C	C	C	C	-	P	P	P	-	C
PURSLANE	C	P	P	C	C	P	N	C	-	C
RADISH, WILD	C	C	C	C	N	C	N	N	-	N
REMORAIDS	-	-	-	C	-	-	-	-	-	-
RUSSIAN THISTLE	P	P	P	C	P	-	N	C	-	C
SHEPHERD'S-PURSE	C	C	C	C	P	C	N	P	-	P
SMARTWEED	-	P	P	C	P	-	-	-	C	-
SOWTHISTLE, ANNUAL	C	C	C	C	P	C	N	C	-	P
SPEEDWELL	-	-	-	-	-	-	-	-	-	-
SPURGE, SPOTTED	N	P	P	C	N	C	N	N	-	P
STARTHISTLE, YELLOW	C	P	P	-	C	N	-	-	-	-
SUNFLOWER, COMMON	C	C	C	C	N	P	N	P	-	N
SWINE CRESS	C	N	N	C	P	C	-	C	-	-
TANSY MUSTARD	C	C	C	C	-	C	N	-	-	P
THISTLE, CANADA	-	-	-	P	N	-	-	-	-	-
THORNAPPLE, CHINESE	-	-	-	C	N	-	N	N	-	C
TURKEY MULLEIN	-	N	N	C	-	-	N	C	-	P
VELVETLEAF	-	-	-	-	P	-	-	-	P	-
WILLOWHERB, PANICLE	-	P	P	C	-	-	-	N	-	P
WOODSORREL, CREEPING	C	P	P	P	N	-	P	N	-	-
GRASSES										
BARLEY, HARE	C	N	N	C	P	C	N	C	-	C
BARNYARDGRASS	C	N	N	C	P	C	P	C	-	C
BERMUDAGRASS(Pt)	N	N	N	C	N	N	N	N	-	C
BERMUDAGRASS(Se)	N	N	N	C	N	N	N	C	-	C
BLUEGRASS, ANNUAL	C	N	N	C	C	P	N	C	-	C
BLUEGRASS, BULBOUS	-	N	N	-	-	-	-	-	-	-
BROME, DOWNY	-	N	N	C	C	C	-	C	-	C
BROME, RIPGUT	-	N	N	C	-	-	-	C	-	C
CANARYGRASS	C	N	N	C	C	C	N	C	-	C
CEREALS, VOLUNTEER	P	N	N	C	N	P	N	C	-	C
CRABGRASS	C	N	N	C	C	C	C	C	P	C
DALLISGRASS(Pt)	N	N	N	P	N	N	C	N	-	N
DALLISGRASS(Se)	N	N	N	C	C	C	C	C	-	N
FESCUE	-	N	N	C	-	-	-	-	-	-
FOXTAIL, YELLOW	C	N	N	C	C	C	-	C	C	C
GOOSEGRASS	C	N	N	C	C	P	C	-	-	C
JOHNSONGRASS(Pt)	N	N	N	P	N	N	N	C	C	C
JOHNSONGRASS(Se)	N	N	N	C	C	N	C	C	C	C
OAT, WILD	C	N	N	C	N	P	N	C	-	C
PANICUM, FALL	C	N	N	C	C	C	C	C	-	C
RABBITFOOT, POLYPOGON	C	N	N	-	C	-	-	-	-	C
RESUEGRASS	-	N	N	C	-	-	-	-	-	-
RYEGRASS, ITALIAN	N	N	N	C	C	P	N	C	-	C
SANDBUR	-	N	N	C	C	C	C	C	-	C
SPRANGLETOP, MEXICAN	N	N	N	C	C	N	N	C	-	C
STINKGRASS	C	N	N	C	C	P	-	C	-	C

Se = SEEDLING, "P" = PERENNIAL, "-" = NO RATINGS, "N" = NO CONTROL, "P" = PARTIAL CONTROL, "C" = CONTROL

Weed Susceptibility Chart

	ORYZALIN	OXADIAZON	OXYFLUORFEN	PARAQUAT	PELARGONIC ACID	PENDIMETHALIN	PHENMEDIPHAM	PRODIAMINE	PROMETRYN
<u>BROADLEAF</u>									
BINDWEED(Pr)	N	P	N	N	N	N	N	P	N
BINDWEED(Se)	P	C	N	P	-	P	P	P	P
CARROT, WILD	N	-	-	P	-	-	-	N	N
CHICKWEED, COMMON	C	N	N	C	C	C	C	C	C
CLOVER	N	N	P	P	-	N	P	N	P
COCKLEBUR	N	-	C	C	-	N	C	N	C
CUDWEED	N	C	N	N	-	N	P	N	P
CURLY DOCK(Pr)	N	N	N	N	N	N	N	N	N
CURLY DOCK(Se)	P	-	C	C	C	C	C	C	C
DAISY, ENGLISH	N	-	-	N	-	-	-	N	N
DANDELION(Pr)	N	N	N	N	N	N	N	N	N
DANDELION(Se)	N	-	G	N	C	N	C	N	C
DODDER	N	-	N	N	-	P	N	C	N
FIDDLENECK	C	C	C	P	C	C	C	C	C
FILAREE	P	C	C	P	P	N	P	N	C
FLEABANE, HAIRY	N	P	P	P	P	N	C	N	C
FLUVELIN	-	-	-	-	-	-	-	-	-
GOOSEFOOT, NETTLELEAF	C	C	C	C	-	C	C	C	C
GROUNDCHERRY	N	C	C	C	-	N	C	N	C
GROUNDSEL, COMMON	N	C	C	C	C	N	C	N	C
HENBIT	C	C	C	C	-	C	C	C	C
HORSETAIL	N	N	N	N	N	N	N	N	N
HORSEWEED, MARESTAIL	N	P	P	P	P	N	C	N	C
KNOTWEED	C	C	P	P	C	C	P	C	C
KOCHIA	C	-	P	C	-	C	P	C	C
KYLINGA, GREEN	N	N	N	-	-	N	-	N	N
LAMBSQUARTERS	C	C	C	C	P	C	C	C	C
LONDON ROCKET	N	C	P	C	C	P	C	N	C
MALLOW, CHEESEWEED	P	C	C	P	C	P	P	N	C
MINERS LETTUCE	-	-	-	C	-	-	-	-	-
MORNINGGLORY, JAPANESE	P	P	C	P	-	N	P	N	C
MUSTARD	N	C	C	C	C	P	C	N	C
NETTLE, BURNING	P	C	C	P	-	N	C	N	C
NIGHTSHADE, BLACK	N	C	C	C	-	N	C	N	C
NIGHTSHADE, HAIRY	N	C	C	C	-	N	C	N	C
NUTSEDGE, PURPLE	N	N	N	N	N	N	N	N	N
NUTSEDGE, YELLOW	N	N	N	N	N	N	N	N	N
ONION, WILD	N	-	N	-	-	-	-	N	N
PEPPERWEED, PERENNIAL	N	N	N	N	N	N	N	N	N
PIGWEEED	C	C	C	C	P	C	C	C	C
PINEAPPLE WEED	N	C	P	P	-	N	P	P	C
PLANTAIN, BUCKHORN(Pr)	N	N	N	N	N	N	N	N	N
PLANTAIN, BUCKHORN(Se)	-	-	-	C	C	-	-	-	C
PRICKLY LETTUCE	N	C	C	P	-	N	C	N	C
PUNCTUREVINE	P	-	C	C	-	P	P	P	P
PURSLANE	C	C	C	C	C	C	C	C	C
RADISH, WILD	N	C	C	C	-	N	C	N	C
REDMAIDS	-	-	-	C	-	-	-	-	-
RUSSIAN THISTLE	P	-	P	C	C	P	-	P	P
SHEPHERD'S-PURSE	N	C	P	P	C	P	C	N	C
SMARTWEED	-	-	-	P	-	-	P	-	-
SOWTHISTLE, ANNUAL	N	C	C	P	-	N	C	N	C
SPEEDWELL	-	-	-	C	-	-	-	-	-
SPURGE, SPOTTED	C	C	N	C	-	P	C	P	-
STARTHISTLE, YELLOW	N	-	C	C	-	N	-	-	C
SUNFLOWER, COMMON	N	-	C	P	-	N	P	N	C
SWINE CRESS	-	-	N	C	-	P	N	-	C
TANSY MUSTARD	N	-	-	P	-	N	C	N	C
THISTLE, CANADA	N	-	-	N	-	-	-	N	N
THORNAPPLE, CHINESE	N	-	P	P	-	N	-	-	P
TURKEY MULLEIN	N	-	P	P	-	N	-	N	-
VELVETLEAF	N	C	-	C	C	N	N	N	-
WILLOWHERB, PANICLE	P	-	C	N	-	-	-	N	-
WOODSORREL, CREEPING	P	C	C	P	P	P	-	P	-
<u>GRASSES</u>									
BARLEY, HARE	C	C	P	P	-	C	N	C	C
BARNYARDGRASS	C	C	P	P	C	C	N	C	P
BERMUDAGRASS(Pr)	N	N	N	N	N	N	N	N	N
BERMUDAGRASS(Se)	C	-	N	P	-	C	N	C	P
BLUEGRASS, ANNUAL	C	C	P	P	C	C	N	C	C
BLUEGRASS, BULBOUS	N	-	-	P	-	-	N	C	N
BROME, DOWNY	C	-	N	P	-	C	N	C	-
BROME, RIPGUT	C	C	-	-	-	C	N	C	P
CANARYGRASS	C	-	P	P	-	C	N	C	C
CEREALS, VOLUNTEER	C	-	N	P	-	C	N	C	P
CRABGRASS	C	C	N	C	-	C	N	C	P
DALLISGRASS(Pr)	N	N	N	N	N	N	N	N	N
DALLISGRASS(Se)	C	-	N	N	-	C	N	C	C
FESCUE	-	-	-	P	-	-	N	-	-
FOXTAIL, YELLOW	C	C	N	C	-	C	N	C	-
GOOSEGRASS	C	C	-	P	-	C	N	C	-
JOHNSONGRASS(Pr)	N	N	N	N	N	N	N	N	N
JOHNSONGRASS(Se)	C	-	N	C	-	C	N	C	N
OAT, WILD	C	C	P	P	-	P	N	P	C
PANICUM, FALL	C	C	N	N	-	C	N	C	-
RABBITFOOT, POLYPOGON	C	-	-	C	C	C	N	C	N
RESCUEGRASS	C	-	-	-	C	N	C	C	-
RYEGRASS, ITALIAN	C	N	N	P	-	C	N	C	P
SANDBUR	P	-	N	P	-	C	N	C	-
SPRANGLETOP, MEXICAN	C	-	N	N	N	C	N	C	N
STINKGRASS	C	-	C	P	-	C	N	C	P

Weed Susceptibility Chart

	PRONAMIDE	PYRAZON	PYRITHOBAC	RIMSULFURON	SETHOXYDIM	SIMAZINE	SULFENTRAZONE	THIAZOPYR	TRICLOPYR	TRIFLURALIN
BROADLEAF										
BINDWEED(Pt)	N	N	N	P	N	N		P	P	P
BINDWEED(Se)	N	N	P	P	N	C		C	C	P
CARROT, WILD	N	-	-	-	N	-		-	C	N
CHICKWEED, COMMON	C	C	N	C	N	C		P	C	C
CLOVER	N	N	N	-	N	C		-	C	N
COCKLEBUR	N	P	C	P	N	C		-	-	N
CUDWEED	N	C	N	C	N	N		C	-	N
CURLY DOCK(Pt)	N	N	N	-	N	N		N	P	N
CURLY DOCK(Se)	C	C	N	C	N	C		-	C	C
DAISY, ENGLISH	N	-	-	-	N	-		-	-	N
DANDELION(Pt)	N	N	N	N	N	N		-	C	N
DANDELION(Se)	N	C	N	P	N	C		-	C	N
DODDER	P	N	N	C	N	N		C	N	C
FOODLENECK	C	C	N	C	-	C		C	-	C
FILAREE	N	P	N	C	N	C		C	P	N
FLEABANE, HAIRY	N	C	N	C	N	C		C	-	N
FLUVELIN	-	-	-	-	N	-		-	-	-
GOOSEFOOT, NETTLELEAF	C	C	C	-	N	C		-	C	C
GROUNDCHERRY	C	C	P	-	N	C ¹		-	C	N
GROUNDSEL, COMMON	N	C	N	C	N	P ¹		C	C	N
HENBIT	C	C	N	C	N	C		-	-	C
HORSETAIL	N	N	N	-	N	N		N	N	N
HORSEWEED, MARESTAIL	N	C	N	-	N	C		C	C	N
KNOTWEED	C	P	C	C	N	C		C	-	C
KOCHIA	C	C	-	C	N	C		-	-	-
KYLLINGA, GREEN	N	-	-	-	N	-		-	-	N
LAMBSQUARTERS	C	C	N	P	N	C		P	C	C
LONDON ROCKET	C	C	C	C	N	C		P	C	N
MALLOW, CHEESEWEED	P	P	N	C	N	P		C	N	N
MINERS LETTUCE	P	C	-	-	N	-		-	-	C
MORNINGGLORY, JAPANESE	-	P	P	P	N	C		-	-	N
MUSTARD	C	C	C	C	N	C		P	C	N
NETTLE, BURNING	C	C	P	-	N	C		C	-	N
NIGHTSHADE, BLACK	C	C	C	P	N	C		P	C	N
NIGHTSHADE, HAIRY	C	C	C	P	N	C		-	C	N
NUTSEDGE, PURPLE	N	N	N	-	N	N		P	N	N
NUTSEDGE, YELLOW	N	N	N	P	N	N		P	N	N
ONION, WILD	-	-	-	-	N	-		-	P	N
PEPPERWEED, PERENNIAL	N	N	N	-	N	N		N	N	N
PIGWEEED	C	P	C	C	N	C		P	C	C
PINEAPPLE WEED	N	P	-	C	N	C		-	-	N
PLANTAIN, BUCKHORN(Pt)	N	N	-	C	N	N		-	P	N
PLANTAIN, BUCKHORN(Se)	N	-	-	C	N	C		-	C	-
PRICKLY LETTUCE	N	C	N	C	N	C		C	C	N
PUNCTUREVINE	N	P	P	C	N	P		-	-	P
PURSLANE	C	C	P	P	N	C		C	-	C
RAIDISH, WILD	P	C	N	C	N	P		-	C	N
REDMAIDS	-	C	-	-	N	-		C	-	-
RUSSIAN THISTLE	P	P	N	P	N	C		-	P	P
SHEPHERD'S-PURSE	C	C	C	C	N	C		C	C	N
SMARTWEED	-	-	-	P	N	C		-	-	-
SOWTHISTLE, ANNUAL	N	C	N	C	N	C		C	C	N
SPEEDWELL	-	C	-	-	N	-		-	-	-
SPURGE, SPOTTED	P	C	N	-	N	N		P	P	P
STARTHISTLE, YELLOW	N	-	-	N	N	C		-	P	N
SUNFLOWER, COMMON	N	P	C	C	N	C		-	C	N
SWINE CRESS	N	C	-	-	N	C		P	-	N
TANSY MUSTARD	C	C	-	C	N	C		-	-	N
THISTLE, CANADA	N	-	-	-	N	N		-	P	N
THORNAPPLE, CHINESE	N	-	P	-	N	-		-	-	N
TURKEY MULLEN	N	-	-	-	N	N		-	-	N
VELVETLEAF	-	-	C	P	N	-		C	-	N
WILLOWHERB, PANICLE	-	-	-	-	N	N		-	-	-
WOODSORREL, CREEPING	-	N	N	-	N	C		C	C	P
GRASSES										
BARLEY, HARE	C	N	N	C	P	C		C	N	C
BARNYARDGRASS	C	N	N	C	C	P		C	N	C
BERMUDAGRASS(Pt)	N	N	N	N	P	N		C	P	N
BERMUDAGRASS(Se)	C	N	N	-	C	P		C	P	C
BLUEGRASS, ANNUAL	C	N	N	C	N	C		C	N	C
BLUEGRASS, BULBOUS	C	N	N	-	N	C		C	N	P
BROME, DOWNY	C	N	N	-	P	C		C	N	C
BROME, RIPGUT	C	N	N	C	C	-		-	N	C
CANARYGRASS	C	N	N	-	C	C		C	N	C
CEREALS, VOLUNTEER	C	N	N	C	C	C		C	N	P
CRABGRASS	C	N	N	P	C	N		C	N	C
DALLISGRASS(Pt)	N	N	N	-	-	N		-	N	N
DALLISGRASS(Se)	C	N	N	-	C	C		-	N	C
FESCUE	-	N	N	-	-	-		-	N	-
FOXTAIL, YELLOW	C	N	N	C	C	C		C	N	C
GOOSEGRASS	C	N	N	C	-	C		C	N	C
JOHNSONGRASS(Pt)	N	N	N	-	C	N		-	N	N
JOHNSONGRASS(Se)	C	N	N	P	C	C		-	N	C
OAT, WILD	C	N	N	P	C	C		-	N	P
PANICUM, FALL ¹	C	N	N	C	-	C		-	N	C
RABBITFOOT, POLYPOGON	-	N	N	C	C	C		C	N	C
RESCUEGRASS	C	N	N	C	C	-		-	N	C
RYEGRASS, ITALIAN	C	N	N	C	C	P		-	N	C
SANDBUR	-	N	N	-	C	C		C	N	C
SPRAGLETOP, MEXICAN	C	N	N	-	C	N		-	N	C
STINKGRASS	C	N	N	P	-	P		-	N	C

Se = SEEDLING, "P" = PERENNIAL, "-" = NO RATINGS, "N" = NO CONTROL, "P" = PARTIAL CONTROL, "C" = CONTROL

Weed Susceptibility Chart

	TRIFLUSULFURON	2,4-D	2,4-OB
<u>BROADLEAF</u>			
BINDWEED(Pr)	P	N	
BINDWEED(Se)	C	N	
CARROT, WILD	-	-	
CHICKWEED, COMMON	P	N	
<u>CLOVER</u>	P	N	
COCKLEBUR	C	C	
CUDWEED	P	P	
CURLY DOCK(Pr)	P	P	
CURLY DOCK(Se)	C	C	
DAISY, ENGLISH	-	-	
DANDELION(Pr)	P	N	
DANDELION(Se)	C	C	
DODDER	N	N	
FIDDLERNECK	P	P	
FLAREE	C	C	
FLEABANE, HAIRY	C	C	
FLUVELIN	-	-	
GOOSEFOOT, NETTLELEAF	C	C	
GROUNDCHERRY	C	C	
GROUNDSEL, COMMON	C	N	
<u>HENBIT</u>	P	P	
HORSETAIL	N	N	
HORSEWEED, MARESTAIL	C	C	
KNOTWEED	P	P	
KOCHIA	C	C	
KYLLINGA, GREEN	-	-	
LAMBSQUARTERS	C	C	
LONDON ROCKET	C	C	
MALLOW, CHEESEWEED	P	P	
MINERS LETTUCE	-	N	
MORNINGGLORY, JAPANESE	C	P	
MUSTARD	C	C	
NETTLE, BURNING	P	P	
NIGHTSHADE, BLACK	C	C	
NIGHTSHADE, HAIRY	C	C	
NUTSEDGE, PURPLE	N	N	
NUTSEDGE, YELLOW	N	N	
ONION, WILD	-	-	
PEPPERWEED, PERENNIAL	P	N	
PIGWEE	P	P	
PINEAPPLE WEED	P	P	
PLANTAIN, BUCKHORN(Pr)	P	P	
PLANTAIN, BUCKHORN(Se)	C	C	
PRICKLY LETTUCE	C	C	
PUNCTUREVINE	C	P	
PURSLANE	C	P	
RADISH, WILD	C	C	
REDMAIDS	-	-	
RUSSIAN THISTLE	C	P	
SHEPHERD'S-PURSE	C	C	
SMARTWEED	P	-	
SOWTHISTLE, ANNUAL	C	C	
SPEEDWELL	-	-	
SPURGE, SPOTTED	P	-	
STARTHISTLE, YELLOW	C	P	
SUNFLOWER, COMMON	C	C	
SWINE CRESS	N	N	
TANSY MUSTARD	C	C	
THISTLE, CANADA	P	N	
THORNAPPLE, CHINESE	C	P	
TURKEY MULLEIN	P	-	
VELVETLEAF	C	-	
WILLOWHERB, PANICLE	C	-	
WOODSORREL, CREEPING	P	-	
<u>GRASSES</u>			
BARLEY, HARE	N	N	
BARNYARDGRASS	N	N	
BERMUDAGRASS(Pr)	N	N	
BERMUDAGRASS(Se)	N	N	
BLUEGRASS, ANNUAL	N	N	
BLUEGRASS, BULBOUS	N	N	
BROME, DOWNY	N	N	
BROME, RIPGUT	N	N	
CANARYGRASS	N	N	
CEREALS, VOLUNTEER	N	N	
CRABGRASS	N	N	
DALLISGRASS(Pr)	N	N	
DALLISGRASS(Se)	N	N	
FESCUE	N	N	
FOXTAIL, YELLOW	N	N	
GOOSEGRASS	N	N	
JOHNSONGRASS(Pr)	N	N	
JOHNSONGRASS(Se)	N	N	
OAT, WILD	N	N	
PANICUM, FALL	N	N	
RABBITFOOT, POLYPOGON	N	N	
RESCUEGRASS	N	N	
RYEGRASS, ITALIAN	N	N	
SANDBUR	N	N	
SPRANGLETOP, MEXICAN	N	N	
STINKGRASS	N	N	

BROADLEAF
 BINDWEED(Pr)
 BINDWEED(Ss)
 CARROT, WILD
 CHICKWEED, COMMON
 CLOVER
 COCKLEBUR
 CUDWEED
 CURLY DOCK(Pr)
 CURLY DOCK(Ss)
 DAISY, ENGLISH
 DANDELION(Pr)
 DANDELION(Ss)
 DODDER
 FIDDLENECK
 FILAREE
 FLEABANE, HAIRY
 FLUVELIN
 GOOSEFOOT, NETTLELEAF
 GROUNDCHERRY
 GROUNDSSEL, COMMON
 HENBIT
 HORSETAIL
 HORSEWEED, MARESTAIL
 KNOTWEED
 KOCHIA
 KYLLINGA, GREEN
 LAMBSQUARTERS
 LONDON ROCKET
 MALLOW, CHEESEWEED
 MINERS LETTUCE
 MORNINGGLORY, JAPANESE
 MUSTARD
 NETTLE, BURNING
 NIGHTSHADE, BLACK
 NIGHTSHADE, HAIRY
 NUTSEDGE, PURPLE
 NUTSEDGE, YELLOW
 ONION, WILD
 PEPPERWEED, PERENNIAL
 PIGWEED
 PINEAPPLE WEED
 PLANTAIN, BUCKHORN(Pr)
 PLANTAIN, BUCKHORN(Ss)
 PRICKLY LETTUCE
 PUNCTUREVINE
 PURSLANE
 RADISH, WILD
 REDMAIDS
 RUSSIAN THISTLE
 SHEPHERD'S-PURSE
 SMARTWEED
 SOWTHISTLE, ANNUAL
 SPEEDWELL
 SPURGE, SPOTTED
 STARTHISTLE, YELLOW
 SUNFLOWER, COMMON
 SWINE CRESS
 TANSY MUSTARD
 THISTLE, CANADA
 THORNAPPLE, CHINESE
 TURKEY MULLEIN
 VELVETLEAF
 WILLOWHERB, PANICLE
 WOODSORREL, CREEPING
 GRASSES
 BARLEY, HARE
 BARNYARDGRASS
 BERMUDAGRASS(Pr)
 BERMUDAGRASS(Ss)
 BLUEGRASS, ANNUAL
 BLUEGRASS, BULBOUS
 BROME, DOWNY
 BROME, RIPGUT
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 FOXTAIL, YELLOW
 GOOSEGRASS
 JOHNSONGRASS(Pr)
 JOHNSONGRASS(Ss)
 OAT, WILD
 PANICUM, FALL
 RABBITFOOT, POLYPOGON
 RESCUEGRASS
 RYEGRASS, ITALIAN
 SANDBUR
 SPRAGLETOP, MEXICAN
 STINKGRASS

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 BINDWEED(Se)
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 CHICKWEED, COMMON
 CLOVER
 COCKLEBUR
 CUDWEED
 CURLY DOCK(Pr)
 CURLY DOCK(Se)
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 DANDELION(Se)
 DODDER
 FIDDLENECK
 FILAREE
 FLEABANE, HAIRY
 FLUVELLIN
 GOOSEFOOT, NETTLELEAF
 GROUNDCHERRY
 GROUNDSSEL, COMMON
 HENBIT
 HORSETAIL
 HORSEWEED, MARESTAIL
 KNOTWEED
 KOCHIA
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